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Alcohol-related Knowledge, Attitude, Belief and Behavior (KABB) Surveys of Alaskan Health Professionals

The Alaska Fetal Alcohol Syndrome Prevention Steering Committee[1]

ABSTRACT

Surveys were conducted to identify alcohol-related and fetal alcohol syndrome training and resource needs of health care providers in the state of Alaska. Surveys were mailed to all obstetrician/gynecologists, pediatricians, family practitioners, and public health nurses in 1992 and 1993. The majority of respondents (95 - 100%) agreed it was their role to address alcohol abuse problems among their patients and their patient's families. The health care providers feeling very or somewhat prepared to deal with alcohol issues were more likely to report that they always refer alcohol-abusing patients to alcohol treatment programs (52% of 158) than those feeling unprepared (24% of 68) (Prevalence ratio 2.2, 95% CI [1.4, 3.5]). The survey results identify a need to facilitate referrals from health care providers. Training to improve provider's level of preparation in dealing with patients in the area of alcohol abuse is indicated and may enhance referrals to treatment services.

INTRODUCTION

Alaska ranked among the top five states in the country for frequent alcohol consumption (> 30 drinks in the past month or 5 or more drinks on one or more occasion in the past month) among women of reproductive age (1). In addition, Alaska had the highest rate of alcohol-related hospitalizations among elderly people in the United States (2). Given the high prevalence of alcohol consumption and alcohol-related morbidity in Alaska, fetal alcohol syndrome (FAS) surveillance and prevention has become a high priority (3).

In an effort to design a comprehensive FAS prevention program, the Alaska FAS Prevention Steering Committee conducted surveys of Alaskan health care providers. The surveys were designed to ascertain each profession's knowledge, attitudes, beliefs, and behaviors (KABBs) concerning alcohol abuse and FAS, and thus identify training and resource needs of the health care providers in the state.

Toll-free Help in Identifying Referral Resources Statewide

The State's Section of Maternal, Child and Family Health (MCFH) offers a toll-free 800 number, called the Healthy Alaskans Information Line, which can assist health care professionals in identifying a wide array of medical, social, and community services to offer their patients. For example, it has information regarding available substance abuse services anywhere in the state. The line can also provide assistance to Alaskan families in need of information regarding health care practitioners who provide maternal and child health services.

MCFH has asked all Alaskan direct health care providers to be a part of the line's database by providing information about their services. If you have not received a program information survey but want to be included, please call the line to request one.

The number for the Healthy Alaskans Information Line is 1-800-478-2221 and operates from 8 am to 9 pm, Monday through Friday.

---

[1] A collaborative effort of the State of Alaska, the Indian Health Service (IHS), and the Centers for Disease Control and Prevention (CDC). Diane Ingle, Sandy Cassidy, Marchelle Kesler, Elaine McKenzie, Karen Pearson, Suzanne Perry, Kaye Saxon, Al Zangri, Sharon Zandman-Zeman, Anita Stevens, Richard Smiley and Drs. Brad Gessner and John Middaugh of the State of Alaska; Jeanne Wolf of the Municipality of Anchorage; Vicki Hild and Dr. James Berner of the IHS; Kathy Perham-Hester of Computer Sciences Corporation; Marvin Bailey, Drs. Carl Li and Grace Egeland of the CDC.

METHODS

Surveys were mailed to all licensed and practicing public health nurses, pediatricians, obstetricians and gynecologists, and family practitioners in the state of Alaska. Postcards noting the recipient's name were sent back separately from the surveys to enable follow-up of non-responders and protect anonymity of respondents.

Survey questions included each profession's perceived role in dealing with issues related to alcohol abuse and to FAS, level of preparation for dealing with alcohol-related problems, knowledge of FAS diagnostic criteria, and the frequency with which each respondent encountered alcohol-related problems. Providers were also asked about their resource needs for dealing with alcohol and FAS-related problems in their practices. Although the majority of questions were on all surveys, the surveys sent to obstetricians and family practitioners focused on obstetric patient care, while the surveys sent to pediatricians and public health nurses focused on pediatric care.

Surveys were mailed to 140 public health nurses, 66 pediatricians, 43 obstetrician/gynecologists (ob/gyns), and 218 family practitioners in 1992 and 1993. After the second mailing, the response rate was 76% (107) for public health nurses, 58% (38) for pediatricians, 67% (29) for ob/gyns, and 61% (132) for family practice physicians.

RESULTS

The majority of health care providers (95-100%) agreed it was their role to address alcohol abuse problems among their patients and their patients' families (Table 1). In addition, the majority agreed that FAS is an identifiable and diagnosable syndrome. A large percentage of public health nurses (88%), ob/gyns (79%), and family practice physicians (81%) agreed that making a diagnosis of FAS can improve treatment plans for the affected child, while only 63% of pediatricians agreed with the statement. Only a small percentage (10-11%) of the health professionals agreed with the statement that discussing alcohol abuse will frighten or anger patients and deter patients from continuing to see the provider.

The level of preparedness in dealing with alcohol abuse issues among patients or parents of pediatric patients, and the frequency of patient referrals to alcohol treatment services varied considerably by professional group. Nearly half of public health nurses (47%) and pediatricians (47%) stated that they felt minimally prepared or unprepared to deal with patients or parents in the area of alcohol abuse. In contrast, only a minority of ob/gyns (11%) and family practice physicians (8%) stated that they felt minimally or unprepared to deal with patients in the area of alcohol abuse. A low percentage of public health nurses (34%) and of pediatricians (19%) reported that they always referred parents of pediatric patients to a drug or alcohol treatment program when they became aware of a substance abuse problem, while over half of ob/gyns (63%) and family practice physicians (64%) stated that they always referred obstetric patients with a known or strongly suspected alcohol abuse problem to alcohol abuse inpatient or outpatient services (Table 1).

Respondents who felt very or somewhat prepared to deal with alcohol issues were more likely than other respondents to report always referring alcohol-abusing patients or parents to alcohol treatment programs: 52% (82) of 158 respondents who felt prepared to deal with alcohol issues versus 24% (16) of 68 who were unprepared reported always referring alcohol-abusing patients or parents to treatment services (Prevalence ratio 2.2, 95% CI [1.4, 3.5]).

IDENTIFICATION OF ALCOHOL ABUSE AMONG OBSTETRIC PATIENTS

While the majority of ob/gyns (97%) and family practice physicians (96%) agreed that identifying prenatal alcohol abuse can improve treatment plans for the pregnancy, the majority (97% and 89%, respectively) disagreed that alcohol abuse is easy to identify. The majority of ob/gyns (96%) and family practice physicians (86%) reported that they always ask obstetric patients if they use alcohol (Table 2). A lower percentage report that they always inform obstetric patients about the dangers of alcohol on the developing fetus (79% and 82%, respectively). Similarly, the majority of family practice physicians (83%) and ob/gyns (96%) reported that they always approached the topic of alcohol abuse with the patients when they knew or strongly suspected an alcohol abuse problem. However, in cases where they delivered infants with known prenatal alcohol exposure, only half of the family practice physicians (46%) and ob/gyns (50%) stated that they always noted alcohol exposure on the birth certificate. When asked whether they agreed that "alcohol abusing pregnant women should be reported to child protective services", 46% of family practice physicians and 52% of ob/gyns agreed.

FAS DIAGNOSIS

The majority of pediatricians (71%) and the minority of family practice physicians (37%) stated that they had ever made a diagnosis of FAS, and over a third of public health nurses (36%) stated they had ever noted FAS in Alaska. Over half of family practice physicians (64%),

Table 1.
Percent of survey respondents* for belief and behavior questions regarding alcohol abuse issues and fetal alcohol syndrome (FAS) by health care provider, Alaska 1992-93.

"Belief/Behavior"	Public Health Nurses (n=107)	Pediatricians (n=38)	Family Practice Physicians (n=132)	Ob/Gyns (n=29)
Agree that it is the role of their profession to address alcohol abuse issues among patients and their families	98	95	97	100
Believe FAS is an identifiable and diagnosable syndrome	94	97	92	90
Believe making a diagnosis of FAS can improve treatment plans for the affected child	88	63	81	79
Believe discussing alcohol abuse will frighten or anger patients/parents and/or deter them from continuing to see the provider	10	11	11	10
Felt minimally prepared or unprepared to deal with patients/parents in the area of alcohol abuse	47	47	8	11
Always referred a patient to alcohol abuse inpatient or outpatient services when they knew or strongly suspected an alcohol abuse problem	34**	19**	64	63

* For any question, the proportion of respondents with missing data was no greater than 8 percent.
** Pediatricians and public health nurses were asked about parents of pediatric patients.

and a third of the pediatricians (34%) stated that they felt uncomfortable making a diagnosis of FAS. All pediatricians and 98% of public health nurses stated that central nervous system dysfunction, facial dysmorphology, and growth retardation were always or sometimes indicative of fetal alcohol syndrome. (Family practice physicians and ob/gyns were not asked about the conditions indicating an FAS diagnosis.)

The term Fetal Alcohol Effects (FAE) was identified as a partial expression of the FAS syndrome in the presence of maternal alcohol history among 93% of the public health nurses, 97% of the pediatricians, and 71% of the family practice physicians. Half of the public health nurses (54%) and family practice physicians (49%) and a fourth of pediatricians (24%) stated that they felt minimally prepared or unprepared to deal with patients or parents concerning problems related to FAS or FAE. Over a third of pediatricians (35%) and of public health nurses (37%) stated that they always approached the topic of prenatal alcohol exposure with the parents or legal guardians of a child with problems associated with prenatal alcohol exposure. When asked whether they agreed that "FAS cases should be reported to [child protective services]", 30% of pediatricians and 18% of public health nurses agreed.

Table 2.
Percent of survey respondents* for belief and behavior questions regarding alcohol abuse among obstetric patients by health care provider, Alaska 1993.

"Belief/Behavior"	Family Practice Physicians (n=132)	Ob/Gyns (n=29)
Always ask obstetric patients if they use alcohol	86	96
Always inform obstetric patients about the dangers of alcohol on the developing fetus	82	79
Ever referred a woman to an alcohol treatment/counseling program	90	90
Always approached the topic of alcohol abuse with the patients seen in the last year with a known or suspected alcohol abuse problem	83	96
Always noted alcohol use on the birth certificate, in cases where alcohol abuse was known or strongly suspected	46	50

* For any question, the proportion of respondents with missing data was no greater than 8 percent.

RESOURCE NEEDS BY PROFESSIONAL GROUP

The majority of family practice physicians requested referral resources for children with FAS and materials or training on identifying FAS. The majority of pediatricians and public health nurses requested lists of resources for parents with alcohol problems and FAS literature for distribution to parents. The majority of ob/gyns also requested these same resources and materials for their patients. In addition, pediatricians requested information on support group referral resources for parents of FAS/FAE children, and public health nurses requested referral resources for children with FAS as well as a registry of specialists for consultation for children diagnosed with FAS.

DISCUSSION

Although the majority of providers believe it is their role to address alcohol abuse issues among their patients, fewer reported that they always refer their alcohol-abusing patients or parents of patients to alcohol treatment services. The survey results identify a need to facilitate referrals from health care providers. Interestingly, the majority (68%) of respondents indicated that a list of resources for individuals with alcohol abuse problems would be helpful to them. A directory of resources may provide a means of increasing physician referrals to treatment services. The difference observed by professional group in the extent to which they always refer individuals to alcohol treatment services may reflect, in part, the fact that obstetrician/gynecologists and family practice physicians were asked specifically about obstetric patients, whereas public health nurses and pediatricians were asked about parents of pediatric patients.

The finding that those who reported feeling prepared to deal with alcohol issues were more likely to always refer alcohol-abusing patients or parents to treatment programs indicate that provider training may enhance referrals.

The vast majority of ob/gyns and family practice physicians reported that they ask obstetric patients if they use alcohol, and inform them about the dangers of alcohol on the developing fetus. These self-reported physician data are supported by the recent results of a survey of mothers of babies born in Alaska during 1990 and 1991 which found that 92% of respondents reported that a doctor or nurse had asked them about their alcohol consumption during their pregnancy (4). Only 77%, however, reported that a doctor or nurse counseled them about the effects of drinking on their baby.

Education regarding the benefits of diagnosing FAS is indicated. Parents of FAS children state the importance of early identification in promoting a constructive environment for interacting with the child. Identification enables parents, teachers and other professionals to shift their perception of the child from one of "willful misconduct" to "handicapped child with potential" (5). Among pediatricians, 37% disagreed with the statement that diagnosing FAS can improve treatment plans for the affected child.

Finally, the results also indicate that birth certificates are unlikely to provide reliable information on the true extent of prenatal alcohol exposure, with only half of ob/gyns and family practice physicians reporting that they always note alcohol exposure on birth certificates for the alcohol-exposed babies they have delivered.

Knowledge, attitude, belief, and behavior surveys are useful tools for identifying the training and resource needs of professional groups who come into contact with high risk populations. These and other data are being used to develop a comprehensive FAS prevention program in the state of Alaska.

Reprint requests should be sent to:
Kathy Perham-Hester, M.S., M.P.H.
State of Alaska, Section of MCFH
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The Alaska Fetal Alcohol Syndrome (FAS) Prevention Steering Committee, which submitted the above featured article, has published other articles on similar topics. A summary of each is given below. In addition, we give summaries of a physician screening guide and book for parents and educators of children affected by alcohol.

Linking multiple data sources in FAS surveillance in Alaska

To better ascertain cases of FAS in Alaska and to determine the prevalence of this problem among Alaska Natives, data from state sources, an Indian Health Service case file and a private pediatric case file were analyzed. A total of 348 persons were identified as potential FAS cases. Medical records were reviewed to see how many met a surveillance definition for FAS. Eighty-three of 323 records available for chart abstraction met the case definition. Ninety percent of these cases were Alaska Native. A minimum FAS prevalence rate for Alaska Natives was 2.1 per 1,000 live births during 1978-1991. (*CDC. Linking multiple data sources in fetal alcohol syndrome surveillance — Alaska. MMWR 1993;42:312-314.*)

Alcohol consumption among women of childbearing age in Alaska

The prevalence of alcohol consumption among women aged 18-44 years in Alaska and their characteristics were obtained through the 1991 Behavioral Risk Factor Surveillance System (BRFSS). Respondents were grouped into three drinking groups: nondrinker, light drinker and heavy drinker. Non-Native women were more likely to report light drinking (41%) than Alaska Native women (17%). The prevalence of heavy drinking among non-Native women (15%) was half that among Alaska Native women (32%). (*CDC. Prevalence and characteristics of alcohol consumption and fetal alcohol syndrome awareness — Alaska, 1991 and 1993. MMWR 1994;43:3-6.*)

Knowledge, attitudes, beliefs, and behaviors about FAS among Alaskan adults

In March 1993, 400 adults (18+ years old) were interviewed by telephone across Alaska to determine their knowledge, attitudes, beliefs and behaviors related to FAS. Knowledge of FAS was defined as correct answers to all seven true/false statements regarding FAS. Although 91% of respondents had heard of FAS, only 41% were knowledgeable about FAS. Forty-three percent of non-Natives were knowledgeable compared with 27% of Alaska Natives who were knowledgeable. The proportion of respondents classified as knowledgeable was higher among respondents who reported they were likely to discuss with a pregnant friend or relative who consumed alcohol the harmful effects of alcohol on the developing fetus (42%) than among those who indicated they would be unlikely to discuss the issue (14%). (*CDC. Prevalence and characteristics of alcohol consumption and fetal alcohol syndrome awareness — Alaska, 1991 and 1993. MMWR 1994;43:3-6.*)

Use of capture-recapture analyses in FAS surveillance in Alaska

Capture-recapture methods were used to estimate the prevalence of FAS among Alaska Natives born 1982-1989. Potential cases were identified through an Indian Health Service (IHS) case file, a private pediatric case file, and Medicaid claims from private physicians. Of 74 Alaska Native children age 3-10 identified as possible cases, 50 were able to be chart-verified as cases of FAS. Observed prevalence rates from these data were 3.1 per 1,000 live births for children born 1982-1985 (age 7-10 years) and 2.0 per 1,000 live births for children born 1986-1989 (3-6 years). Capture-recapture analyses were conducted using cases identified by IHS and private physicians. These analyses estimated a prevalence of the syndrome of 3.8 per 1,000 live births for children born 1982-1985, and 3.1 per 1,000 live births for children born 1986-1989. Based on the capture-recapture predicted number of cases, the IHS case file ascertained a greater percentage of cases among the older cohort (75%) than among the younger cohort (56%). These data illustrate the use of capture-recapture analyses in identifying the extent to which observed trends in rates may reflect differences in case ascertainment over time (or by birth cohort). (*Egeland GM, Perham-Hester KA, Hook EB. Use of Capture-Recapture Analyses in Fetal Alcohol Syndrome Surveillance in Alaska. Am J Epidemiol, 1995;141:335-41*).

Reprints of the above articles may be requested by writing to the Alaska FAS Prevention Project, 1231 Gambell St., Anchorage, Alaska 99501 or by calling (907) 278-2990. Other resources follow.

A Screening Guide for Fetal Alcohol Syndrome by Sterling K. Clarren, M.D. and Susan J. Astley, Ph.D. (1993). (Sponsored by US Dept. of Health and Human Services, US Public Health Service, National Centers for Disease control and Prevention, Cooperative Agreement U59-CCU006992-2 and the Washington State Dept. of Health, Parent-Child Health Services.)

Dr. Clarren, renowned dysmorphologist and director of the Fetal Alcohol Syndrome Clinic at the University of Washington Child Development and Mental Retardation Center, has delimited the key components to screening children for FAS in a clinical setting. Complete with photographic examples of facial abnormalities consistent with FAS and the standards for facial measurements, this manual is a good resource for any physician unaware of the standard criteria for assigning an FAS diagnosis.

For a copy, you may contact the Alaska FAS Prevention Project at the address and phone number given above.

Fantastic Antone Succeeds! Experiences in Educating Children with Fetal Alcohol Syndrome Edited by Judith Kleinfeld and Siobhan Wescott (1993).

For the first time in a book, parents, teachers, and social workers present the successes of children with fetal alcohol syndrome or effect. The stories tell of the common problems experienced by these children—difficulty concentrating in school, kinesthetic hypersensitivity, trouble with telling time, etc.—and how the children and adults found innovative solutions. *Fantastic Antone Succeeds!* offers the interventions to help children with FAS or FAE avoid many of the more serious problems later in life. The earlier the intervention, the better.

You can order *Fantastic Antone Succeeds!* through its publisher for $20.00 paper or $30.00 cloth with a $3.00 shipping and handling charge. Write to: University of Alaska Press, 1st floor Gruening Bldg. - UAF, Fairbanks, Alaska 99775; or phone (907) 474-6389.

National Organization on Fetal Alcohol Syndrome (NOFAS):	(800) 66-NOFAS
National Clearinghouse for Alcohol and Drug Information US Dept. of Health and Human Services, Public Health Service:	(800) 729-6686

Traumatic Head and Spinal Cord Injuries in Alaska (1991-1993)

Sam Warren, AB(1)
Martha Moore, MS(1)
Mark S. Johnson, MPA(1)

ABSTRACT

Traumatic head and spinal cord injuries (SCI) are serious events, often leading to severe disability or death. Because these injuries are experienced most often by younger, more active people, they are associated with some of the most enduring physical, emotional, and financial costs. In order to describe several demographic and epidemiologic characteristics of traumatic head and spinal cord injuries in Alaska, a retrospective study was conducted using information collected from the Alaska Trauma Registry (ATR) for hospital trauma admissions in 1991 through 1993. Hospitalized traumatic head injury were identified in 2178 cases, for an average annual incidence rate of 129.5 per 100,000 population. For hospitalized traumatic SCI, 139 cases were identified, for an average annual incidence rate of 8.3 per 100,000 population. To analyze and assess the incidence of these injuries in Alaska, the following injury characteristics were explored through the Alaska Trauma Registry: demographics, mechanism of injury, work relatedness, use of safety equipment, involvement of alcohol, and discharge status.

INTRODUCTION

Injury remains the nation's leading killer of people younger than 44 years of age, causing more deaths in the 1-34 age group than all diseases combined. About 80,000 new cases of permanently disabling injury to the brain or spinal cord are reported each year in the United States (1). Most, if not all, of these injuries are potentially preventable.

The first key to injury prevention is injury surveillance. Alaska is unique in that it is one of a small number of states with a surveillance system in place which collects trauma information from every hospital in the state. The Alaska Trauma Registry is a database of up to 144 data elements collected for every trauma patient, as defined by ICD-9-CM injury code, who is admitted to a hospital, transferred to a higher level of hospital care, or declared dead in a hospital emergency department. The data elements are collected by each hospital's designated trauma registrar, usually an emergency department nurse or medical records person. The data are entered onto a worksheet which includes information on patient demographics, circumstances of injury, prehospital and in-hospital response times and treatment, severity of injury, diagnosis, length of hospital stay, cost of hospitalization, and discharge condition.

The confidentiality of Trauma Registry data is protected under Alaska Statute 18.23.010-070. Under that statute, data acquired by a health care review organization, in the exercise of its duties or functions, is not subject to subpoena or discovery. The Trauma Registry Review Committee, comprised of at least 75% health care providers, serves as this organization and meets three times a year to guide trauma registry development and to review data. In addition, all trauma registry personnel sign a confidentiality statement. There are no patient identifiers. Physicians, hospitals, clinics, and ambulance services are coded during data entry. Information is released to requesting agencies in aggregate form.

Data collected through the Alaska Trauma Registry can help hospital staffs and prehospital emergency medical services by providing valuable feedback to analyze and assess the quality and appropriateness of treatment, as well as to determine needs for training, personnel, equipment, and funding. Information provided by the registry is also useful to federal, state, and local governments and other agencies when planning and evaluating injury prevention programs, public education, EMS training, and legislative action.

Efforts aimed at prevention of head and spinal cord injuries are complicated by a number of factors unique to Alaska. Alaska's size (591,000 square miles), uneven population distribution, relative inaccessibility to villages and rural areas, and geographical complexity pose certain obstacles to health and medical care providers and injury prevention specialists not regularly encountered elsewhere in the United States.

---

(1) Emergency Medical Services Section, Division of Public Health, Alaska Department of Health and Social Services, P.O. Box 110616, Juneau, AK 99811-0616

The diversity of injury settings in Alaska amplifies the need for a complete system of injury surveillance capable of describing many injury characteristics in a regionally specific manner, as well as within the state as a whole. The following is a descriptive analysis and assessment of head and spinal cord injury in Alaska, for the years 1991 through 1993, with respect to demographics, mechanisms of injury, work relatedness, use of safety equipment, involvement of alcohol, and discharge status.

MATERIALS AND METHODS

Surveillance-

Head injury was defined as any fracture of the skull, intracranial injury, or injury to any of the cranial nerves as defined by the following ICD-9-CM codes: 800.00-800.99 - *fracture of vault of skull;* 801.00-801.99 - *fracture of base of skull;* 802.00-802.99 - *fracture of face bones;* 803.00-803.99 - *other and unqualified skull fractures;* 804.00-804.99 - *multiple fractures involving skull or face with other bones;* 850.00-850.99 - *concussion;* 851.00-851.99 - *cerebral laceration and contusion;* 852.00-852.99 - *subarachnoid, subdural, and extradural hemorrhage, following injury;* 853.00-853.99 - *other and unspecified intracranial hemorrhage following injury;* 854.00-854.99 - *intracranial injury of other and unspecified nature;* 950.00-950.99 - *injury to optic nerve and pathways; and* 951.00-951.99 - *injury to other cranial nerve(s).*

Spinal cord injury was defined as a lesion of the spinal cord resulting in any motor or sensory deficit or pelvic visceral dysfunction, temporary or permanent, as defined by the following ICD-9-CM codes: 806.00-806.99 - *fracture of vertebral column with spinal cord injury;* and 952.00-953.99 - *spinal cord injury without evidence of spinal bone injury.* Lesions occurring to the cervical spine are given the prefix quadri- while those occurring at the thoracic, lumbar, and sacral levels are given the prefix para-. Injuries specified as "complete" by ICD-9-CM codes are referred to as having complete quadriplegia or paraplegia whereas injuries diagnosed with the other ICD-9-CM codes are considered as having "incomplete" paraparesis or quadriparesis.

Cases were collected from the Alaska Trauma Registry (ATR) by the specified ICD-9-CM codes for 1991 through 1993. Data collection for 1993 was not yet complete at the time of study. Based on the number of injuries reported to the ATR in 1992, an estimated five percent of the total expected 1993 trauma cases were still outstanding. To adjust for the missing data, the total period of study was regarded as 2.95 years for purpose of analysis. Head injury cases were collected and analyzed separately from spinal cord injuries. The following data elements were gathered for analysis: age, gender, race, scene region, residence, external cause of injury code (ICD-9-CM "E" code specifying mechanism of injury), injury description, safety equipment use, work relatedness, occupation, alcohol documentation, blood alcohol level, hospital charges, and discharge status. The reported national rates for traumatic head and spinal cord injury were obtained from the National Center for Health Statistics, U.S. Centers for Disease Control and Prevention, using estimates derived from the National Hospital Discharge Survey of 1992, with the identical definitions based on ICD-9-CM injury codes used in this study.

Race was specified only as non-Native or Native, since no other minority population exceeds 5 percent of the total population in Alaska. Regional breakdowns were gathered in reference to those 14 regions/subregions recognized by Alaska's Emergency Medical Services (EMS) Section. Alcohol documentation refers to those cases in which blood alcohol tested positive or there was documentation contained within the medical record implicating the involvement of alcohol with the injury. Hospital charges reported are a gross underestimate of total cost for a particular injury as they do not include individual physicians' fees, prehospital care and transportation, short and long term rehabilitation and social service costs, or loss of future earning potential.

Annual incidence rates were calculated per 100,000 population using the 1991 estimates from the most recently published Alaska Population Overview published by the Alaska Department of Labor (2).

The computer software supporting the ATR is the Hospital Trauma Register Program developed by Richard Cales, MD, FACEP, formerly the Chairman of the Committee on Trauma of the American College of Emergency Physicians. Chi-square tests used to assess statistical significance were performed by STATPAC GOLD (3).

Sensitivity of Surveillance-

The National Institute for Occupational Safety and Health (NIOSH), Alaska Activity office in Anchorage is currently involved in a validation study to assess the ATR's sensitivity as a surveillance system. This study identifies trauma cases from each hospital's on-site records and compares the total number of identified cases for a 30 day period with the total number of cases in that period collected by the ATR. At each respective hospital, the NIOSH study ascertains trauma cases through the identification of data elements different from those which qualify a case for inclusion in the ATR. To date, sensitivity of surveillance has been assessed in the two largest of the 25 acute care hospitals in Alaska — Providence Hospital and Alaska Regional Hospital in Anchorage. At Providence Hospital, where the ATR identifies cases by ICD-9-CM injury code and by ED log, NIOSH identified trauma patients by ICD-9-CM external cause coding ("E" code) and found the ATR to have missed one case out of a total of 62 cases for that month. At Alaska Regional Hospital, where ATR identifies cases by ICD-9-CM injury code and by death book, NIOSH identified cases by ED log and found no cases that were missed by the ATR out of a total of 35 cases for that month (4). Because this validation study is not yet complete, these findings are not necessarily an accurate index of sensitivity for the registry as a whole. However, given that between 30 and 35 percent of the ATR's total data comes from these two urban hospitals (as sites of first admittance and referral facilities for trauma transfers), this information is an indicator of the ATR's surveillance sensitivity.

The ATR does not collect data for injury patients seen in the emergency department and released without being admitted to the hospital. Thus, no surveillance of minor head injuries such as mild concussions was included. Trauma deaths not transported to a hospital are available from the Alaska Section of Vital Statistics, Division of Public Health. However, the only ICD-9-CM code recorded for injury deaths on death certificates at the time of this study was the external cause of injury code ("E" code), thus making traumatic head and spinal cord injuries impossible to ascertain retrospectively looking at these certificates. During calendar year 1995, the Section of Vital Statistics plans to implement multiple cause of death coding, which should make this type of information (injury diagnosis codes) available for future studies.

RESULTS

Demographics-

For the period of January 1991 through December 1993, a total of 2178 hospitalized traumatic head injuries were identified for an average annual incidence rate of 129.5 per 100,000, and 139 hospitalized traumatic spinal cord injuries were identified for an average annual incidence rate of 8.3 per 100,000. Comparison of these rates to other population-based rates is difficult because of variations in injury definitions and case ascertainment. A Rhode Island count of head injuries published in 1986 showed a rate of 152 per 100,000 population among hospital admissions (5). Published literature on annual incidence rates for hospitalized spinal cord injury range from 2.4 to 5.0 per 100,000 (6). Using the identical injury definitions, the estimated national incidence rates from the National Hospital Discharge Survey for traumatic head and spinal cord injury are 155.4 and 7.9 respectively (7).

Nineteen (19) trauma victims sustained both head injury and SCI during the same event. There were 194 (8.9%) head injury patients and 35 (25.2%) SCI patients who at hospital discharge were predicted never to return to their pre-injury level of function. The average hospital charge for traumatic head injury was $15,055.00; which did not include individual doctors fees, prehospital care and transportation, short and long term rehabilitation, social services, or loss of future earning potential. The equivalent figure for traumatic SCI was $26,567.00. The annual incidence rate of hospitalized traumatic head injury among Native Alaskans was 1.8 times that of the non-Native population (Table 4) ($p < .001$). Native Alaskan incidence of traumatic spinal cord injury in the patient population was 1.3 times the non-Native rate (Table 5). Patients whose place of residence was outside the State of Alaska comprised 5.1% (111 cases) of recorded head injuries and 13.7% (19 cases) of recorded SCIs.

The region specific annual incidence rates are reported in Table 1. In the Anchorage subregion, where approximately 46% of the State's population resides, the rates for hospitalized traumatic head and SCI were 91.3 and 4.9 per 100,000. The highest incidence rates for both traumatic head and spinal cord injuries were shared by the same two regions, Copper River and Bristol Bay. The Copper River patient population experienced a head injury rate almost twice the estimated national rate and

Table 1.

Frequency and annual incidence rates of hospitalized traumatic head and spinal cord injuries, by EMS region/subregion (1991-1993). *

Region/Sub-Region (population)	HEAD No. Annual Rate	SCI No. Annual Rate
Aleut\Prib (12,340)	55 - 151.1	7 - 19.2
Anchorage (237,216)	639 - 91.3	34 - 4.9
Bristol Bay (7,292)	43 - 199.9	8 - 37.2
Copper River (2,801)	25 - 302.6	5 - 60.5
Interior (94,310)	447 - 160.7	15 - 5.4
Kenai Pen (42,242)	198 - 158.9	26 - 20.9
Kodiak (13,959)	51 - 123.8	3 - 7.3
Mat-Su (40,494)	207 - 173.3	11 - 9.2
Northwest Arctic (6,303)	31 - 166.7	3 - 16.1

* Annual rates are per 100,000 population.

a spinal cord injury rate over seven times the estimated national spinal cord injury rate. The Bristol Bay patient population also sustained head injuries at a rate well above the estimated national rate and spinal cord injuries at a rate of almost five times that reported nationally.

Of the spinal cord injuries identified, 1 (0.7%) case involved complete quadriplegia and 8 (5.8%) cases involved complete paraplegia. Sixty-one (43.9%) of the cases were associated with cervical SCI and 94 (67.6%) of the cases involved other levels of the spinal cord. These percentages show that some cases involved lesions at both the cervical and a more inferior level of the spinal cord.

Ages ranged from 0 to 107 years for head injury and 11 to 85 years for SCI. The median patient age was 27 years for head injury and 33 years for SCI. Both injuries tended to occur in the younger population. Greater than 68% of the traumatic head injuries identified were people younger than 35 years of age, compared to 62% of the total population in this age group. Greater than 55% of the SCIs identified were within the 15-34 age range, which includes 34% of the total population.

Males accounted for 72.5% of all head injury cases and had higher annual incidence rates than females for all age groups observed (Table 2). Both male and female annual incidence rates were highest in the 85+ age group. The greatest number of traumatic head injuries identified for males and females was in the 15-24 year age range, 412 and 135 respectively. The 15-24 age group also accounted for the highest annual incidence rates of hospitalized traumatic head injury in both sexes for those victims under age 75, 312.5 cases per 100,000 males and 122.2 cases per 100,000 females.

Table 2.
Frequency and annual incidence rates of hospitalized traumatic head injury in Alaska, by age and sex (1991-1993)*

	Sex						
	Male		Female		Unk	Total	
Age	No.	Annual Rate	No.	Annual Rate	No.	No.	Annual Rate
0-4	89	102.9	58	70.1	0	147	86.9
5-14	189	125.7	86	61.1	0	275	94.5
15-24	412	312.5	135	122.2	0	547	225.8
25-34	404	230.8	120	74.1	1	525	155.8
35-44	237	139.6	89	59.7	0	326	102.3
45-54	124	137.0	33	43.5	0	157	94.4
55-64	57	121.8	32	78.1	0	89	101.4
65-74	41	172.3	24	96.5	0	65	133.6
75-84	20	262.5	15	155.1	0	35	202.4
85+	7	467.1	5	202.5	0	12	302.4
Total	1580	178.8	597	74.7	1	2178	129.5

* Annual rates are per 100,000 population.

Males accounted for 83.5% of traumatic spinal cord injuries and had higher annual incidence rates than females in all age groups observed under the age of 65 (Table 3). The total case counts for age groups above 65 are too low to provide any solid information about incidence in these age groups. Given this, the overall annual incidence rate for hospitalized traumatic SCI was highest in the 25-34 age range for both genders.

Table 3.
Frequency and annual incidence rates of hospitalized traumatic spinal cord injury in Alaska, by age and sex (1991-1993)*

	Male		Female		Total	
Age (yrs)	No.	Annual Rate	No.	Annual Rate	No.	Annual Rate
0-4	0	0	0	0	0	0
5-14	3	2.0	1	0.7	4	1.4
15-24	23	17.4	6	5.4	29	12.0
25-34	42	24.0	6	3.7	48	14.2
35-44	28	16.5	3	2.0	31	9.7
45-54	8	8.8	2	2.6	10	6.0
55-64	9	19.2	1	2.4	10	11.4
65-74	1	4.2	3	12.1	4	8.2
75-84	1	13.1	1	10.3	2	11.6
85+	1	66.7	0	0	1	25.2
Total	116	13.1	23	2.9	139	8.3

* Annual rates are per 100,000 population.

Overall, motor vehicle crashes (involving occupants) and falls were the two leading mechanisms of injury associated with traumatic head injury in Alaska requiring hospitalization, followed by assault and off-road vehicle crashes (Table 4). Assault was the leading cause of hospitalized traumatic head injury in the Native Alaskan population. Native Alaskan patients were 4.1 times more likely to sustain head injury due to assault than non-Natives (p<.001) and 6.8 times more likely to sustain head injury (p<.001) from an off-road vehicle (ATV or snow machine) crash. Consistent with the overall leading mechanism of traumatic head injury, motor vehicle crashes were the leading cause of injury in the non-Native population.

The overall leading mechanisms of injury associated with hospitalized trau-

Table 4.
Frequency and annual incidence rates of hospitalized traumatic head injury in Alaska, by mechanism of injury and race (1991-1993)*+

	Race						
	Non- Native		Native		Unknown	Total	
Mechanism of Injury	No.	Annual Rate	No.	Annual Rate	No.	No.	Annual Rate
motor vehicle (occupant)	417	30.4	76	30.1	35	528	32.5
falls	348	25.4	88	34.8	20	456	28.1
assault	212	15.5	159	62.9	13	384	23.7
off-road vehicle (snowmach/ATV)	71	5.2	89	35.2	3	163	10.0
bicycle	57	4.2	27	10.7	6	90	5.5
pedestrian	46	3.4	32	12.7	0	78	4.8
motorcycle	58	4.2	5	2.0	6	69	4.3
sport/rec	52	3.8	8	3.2	1	61	3.8
water trnspt	43	3.1	6	2.4	2	51	3.1
air trnspt	41	3.0	5	2.0	0	46	2.8
self inflict	21	1.5	4	1.6	1	26	1.6
playgrd falls	11	0.8	10	4.0	2	23	1.4
sledding	19	1.4	1	0.4	1	21	1.3
firearm	7	0.5	11	4.4	1	19	1.2
other	114	8.3	36	14.2	13	163	9.9
Total	1517	110.7	557	220.3	104	2178	134.0

* annual incidence rates per 100,000 population
+ there were 104 head injury cases where race was not collected

Table 5.
Frequency and annual incidence rates of hospitalized traumatic spinal cord injury in Alaska, by mechanism of injury and race (1991-1993)*+

	Race						
	Non-Native		Native		Unknown	Total	
Mechanism of Injury	No.	Annual Rate	No.	Annual Rate	No.	No.	Annual Rate
motor vehicle (occupant)	35	2.9	6	2.4	3	44	2.7
falls	23	1.9	6	2.4	1	30	1.8
off-road vehicle (snow mach/ATV)	13	1.1	7	2.8	0	20	1.2
air transport	10	0.8	0	0	0	10	0.6
assault transport	3	0.2	3	1.2	0	6	0.4
water	5	0.4	1	0.4	0	6	0.4
motorcycle	4	0.3	0	0	0	4	0.2
pedestrian	1	0.1	2	0.8	0	3	0.2
firearm	2	0.2	0	0	0	2	0.1
bicycle	2	0.2	0	0	0	2	0.1
sledding	1	0.1	0	0	1	2	0.1
other	8	0.7	1	0.4	1	10	0.6
total	107	8.7	26	10.3	6	139	8.6

* annual incidence rates per 100,000
+ there were 6 SCI cases where race was not collected

matic SCI in Alaska were motor vehicle (occupant) crashes, falls, off-road vehicle crashes, and air transport crashes (Table 5). Motor vehicle crashes were also the leading mechanism of these injuries in the non-Native population, whereas off-road vehicles were the number one mechanism of SCI within the Native Alaskan population with an annual rate 2.5 times the off-road vehicle SCI rate in the non-Native population (p<.005).

Table 6 lists the leading mechanisms of injury for each respective region for traumatic head and spinal cord injuries. The overall leading mechanisms for the state are reflected in observing those mechanisms of higher incidence within the more densely populated regions such as Anchorage, Fairbanks (Interior), Southeast Alaska, Matanuska-Susitna Borough, and the Kenai Peninsula. Within many of the other, more rural regions of the state, however, leading injury mechanisms differ, keeping somewhat consistent with the occupations and lifestyles of different geographical areas. Examples are the high incidence of water transportation associated head and spinal cord injuries in the Aleutian/Pribilof Islands and the high incidence of ATV and snow machine associated head injuries in the Bristol Bay region.

Of the 19 patients identified with both head and spinal cord injuries, nine (47.4%) were motor vehicle traffic occupants (5 drivers and 4 passengers), two (10.5%) were all-terrain vehicle drivers, two (10.5%) were motorcyclists, and there was one each involving a snowmachine, airplane, water transport, fall, assault, and struck by an object.

Of the 56 head or spinal cord injuries related to water transport: 25 (44.6%) were caused by

Table 6.
Top mechanisms of hospitalized traumatic head injury and hospitalized traumatic spinal cord injury in Alaska, by EMS region/subregion, in percent of each region's total cases (1991-1993)*

Region/Subregion	Mechanisms of Injury*+ Traumatic Head Injury % of region's total		Traumatic SCI % of region's total	
Aleut/Prib	N=55		N=7	
	water transp	30.9	water transp	42.9
	air transp	21.8		
	assault	16.4		
	falls	10.9		
	motor veh	5.5		
Anchorage	N=639		N=34	
	motor veh	25.5	falls	38.2
	falls	25.5	motor veh	17.6
	assault	20.8	assault	11.8
	pedestrian	7.7	air transp	5.9
	bike	5.2	firearm	5.9
	motorcycle	3.1	bike	5.9
	self inflict	1.4		
	sledding	1.1		
Bristol Bay	N=43		N=8	
	ATV	27.9	air transp	25
	snow machine	14.0		
	falls	14.0		
	air transp	11.6		
	water transp	9.3		
	assault	9.3		
	motor veh	7.0		
Copper River	N=25		N=5	
	motor veh	36.0	snow machine	40.0
	falls	16.0		
	assault	12.0		
	snow machine	8.0		
Interior	N=447		N=15	
	falls	25.3	Motor veh	46.7
	motor veh	24.4	ATV	26.7
	assault	17.3	snow machine	13.3
	bike	4.9		
	snow machine	3.6		
	motorcycle	2.9		
	ATV	1.8		
	self inflict	1.1		
	sledding	1.1		
	playgrd fall	1.1		
Kenai Pen	N=198		N=26	
	motor veh	34.2	motor veh	57.5
	falls	17.7	falls	19.2
	assault	10.1	motorcycle	7.7
	motorcycle	8.1	air transp	7.7
	ATV	4.5		
	bike	3.0		
	pedestrian	3.0		
	sledding	3.0		
	snow machine	2.5		
	air transp	2.5		
	self inflict	2.0		
	animal ride	1.5		
Kodiak	N=51		N=3	
	assault	27.5	no mechanism associated with >1 case	
	ATV	15.7		
	falls	13.7		

(continued)

	water transp	15.7		
	motor veh	9.8		
	self inflict	3.9		
	air transp	3.9		
Mat-Su	N=207		N=11	
	motor veh	44.0	motor veh	45.5
	falls	16.9	ATV	18.2
	assault	9.2		
	ATV	6.3		
	motorcycle	4.3		
	snow machine	4.3		
	pedestrian	1.9		
	bike	1.5		
	air transp	1.4		
Northwest Arctic	N=31		N=3	
	ATV	22.6	no mechanism associated with >1 case	
	assault	22.6		
	snow machine	16.1		
	falls	6.5		
	self inflict	6.5		
North Slope	N=34		N=3	
	assault	32.4	no mechanism associated with >1 case	
	snow machine	17.6		
	ATV	14.7		
	motor veh	5.9		
	pedestrian	5.9		
Norton Sound	N=47		N=0	
	falls	17.0	0 cases	
	snow machine	17.0		
	assault	17.0		
	ATV	12.8		
	air transp	10.6		
	motor veh	4.3		
	bike	4.3		
PWS	N=39		N=1	
	motor veh	25.6	no mechanism associated with >1 case	
	falls	25.6		
	assault	10.3		
	air transp	7.7		
SE AK	N=265		N=14	
	falls	20.9	motor veh	35.7
	motor veh	20.5	falls	28.6
	assault	18.3		
	bike	8.4		
	water transp	4.6		
	pedestrian	3.8		
	motorcycle	2.7		
	ATV	2.3		
	playgrd fall	2.7		
	machinery	1.1		
YKHC	N=77		N=7	
	assault	28.6	ATV	42.9
	snow machine	24.7		
	falls	13.0		
	ATV	10.4		
	firearm	5.2		
	motorcycle	2.6		
	dogbite	2.6		
	motor veh	2.6		

* mechanisms of injury associated with 1 case or < 1% of a region's total cases are not listed.

+ ATV and snow machine are reported independently in this table rather than being collectively considered off-road vehicle

falls; 19 (34%) were caused by being struck by an object; 6 (10.7%) involved collisions; 2 (3.6%) involved explosions; 2 (3.6%) were caused by being caught in a winch; 1 by equipment failure (2%); and 1 (2%) unknown.

Of the head injuries identified, 170 (7.8%) were work-related. There also were 23 (16.5%) SCIs recorded as work-related. The overall average percentage of occupational related trauma cases in the ATR is close to 13%. The most commonly involved occupations in work-related traumatic head and spinal cord injuries are shown in Table 7. Fishing, construction, military, and logging occupations were responsible for the highest number of work-related traumatic head injuries. Work-related traumatic spinal cord injuries were spread fairly evenly over several occupations. Only 122 of the 170 work-related traumatic head injuries in the ATR included information on the precise occupation involved. Likewise, in only 18 of 23 work-related SCIs were specific occupations reported. This gap in data collection makes it difficult to determine annual incidence rates for these injuries within individual occupations.

Among the 170 work-related head injuries, 39 (23%) were patients whose resident address was outside the State of Alaska. Seven of the 23 work-related SCI's (30%) were sustained by non-residents.

Of the 2178 traumatic head injuries, there were 885 for which the use of safety equipment was considered applicable. Of these, 660 (74.6%) cases actually reported the use or nonuse of appropriate safety equipment. Of these 660 cases, 70.2% of patients were not using the appropriate safety protection. Of motorcycle, off-road vehicle, and bicycle incidents leading to traumatic head injury, where safety equipment

Table 7.
Occupations most commonly reported in work-related hospitalized traumatic head and spinal cord injuries in Alaska (1991-1993)

Occupation	Traumatic HeadInjury No.	Traumatic SCI No.
logging	14	2
fishing	22	3
mining	2	0
oil/gas	5	0
construction	20	3
air transportation	8	2
land transportation	3	1
water transportation	4	1
services	12	2
manufacturing	1	0
wood processing	1	0
fish processing	8	2
military	16	2
other	6	0
unknown	48	5
Total	170	23

information was available, 84.3% of all victims were not wearing helmets (Table 8). Of the 56 cases of traumatic spinal cord injury where safety equipment was both applicable and use or non-use known, 62.5% of patients were not using appropriate safety equipment at the time of injury.

Of the 2178 head injuries in the database, 583 (27%) had some documentation of alcohol involvement. The remaining 1595 cases (73%) had no documentation of alcohol involvement. Of the 139 traumatic SCI cases, 43 cases (31%) contained some documentation of alcohol involvement, either by a blood alcohol test, or by other indications in the medical record that alcohol was involved. The remaining 96 cases (69%) had no evidence of alcohol involvement.

There were 490 victims of traumatic head injury and 31 victims of traumatic SCI whose blood alcohol levels were tested. The results of these tests are listed in Table 9.

For both head injury and SCI, alcohol involvement was documented over 1.9 times more often when victims were Native Alaskan than when victims were non-Native ($p < .001$ and .005, respectively).

Table 8.
Frequency of hospitalized traumatic head injuries related to motor vehicles, off-road vehicles (snow machine and ATV), and bicycles in which the victim was not using appropriate safety equipment (1991-1993).

	Safety Equipment				
Mechanism of Injury	Not Relevant No.	Relevant and Known No.	None Used No.	None Used Percent	Use Unknown No.
motor vehicle	10	411	269	65.5%	107
motorcycle	0	62	49	79.0%	7
off-road vehicle	0	95	79	83.2%	68
bicycle	0	66	60	90.9	24
airplane	1	26	6	23.1	19
Total	11	660	463	70.2%	225

Table 9.
Results of blood alcohol tests administered on victims of traumatic head and spinal cord injuries in Alaska (1991-1993).

	Traumatic Head Injury		Traumatic SCI	
Blood Alcohol Level (mg/dl)	No.	% of No. tested	No.	% of No. tested
000	99	20.2	6	19.4
001-050	28	5.7	0	0.0
051-099	37	7.6	1	3.2
100-250	196	40.0	14	45.2
251-500	127	25.9	10	32.3
500+	3	0.6	0	0.0
Total Tested	490		31	

Table 10.
Discharge status of hospitalized traumatic head injury and spinal cord injury patients in Alaska (1991-1993).

	HEAD	SCI
Home	1572	65
Left Against Medical Advice	18	1
Transferred to Other Acute Care Facility	361	46
Transferred to Inpatient Rehab	76	21
Transferred to Skilled Nursing	25	0
Transferred to Res. Facility	6	1
Expired	120	5
Total	2178	139

Table 10 lists the disposition of the head and spinal cord injury patients upon discharge from the initial hospital. Of the 2,178 head injury patients, 16.5% were transferred to another acute care hospital, almost 5% were transferred to a rehabilitation or skilled nursing care facility, and 120 patients (5.5%) died. Seventy-two percent were discharged home. Of the 139 spinal cord injury patients, 33% were transferred to an acute care facility, 15.8% to long term care, five (3.4%) died, and 46.8% were discharged home.

DISCUSSION

Data from the Alaska Trauma registry shows that the incidence of traumatic head and spinal cord injuries in Alaska are comparable to national incidence rates from the National Center for Health Statistics. However, rates among Native Alaskans and rates in some regions are much higher than the national rates.

More consistent and reliable reporting of the use of safety equipment and the involvement of alcohol would assist injury prevention planners and others to determine appropriate strategies to prevent many of these types of injuries in the future.

This study shows that the Alaska Trauma Registry can be a useful tool for injury surveillance in Alaska. Analyzing the Alaska rates by region/subregion, sex, race, mechanism of injury, work-relatedness, use of safety equipment, involvement of alcohol, and discharge status reveals some distinct patterns which can be used by injury prevention program planners, elected officials, and others to design and evaluate traumatic head and spinal cord injury prevention strategies.

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DIETARY INTAKES OF PARTICIPANTS IN THE IDITASPORT HUMAN POWERED ULTRA-MARATHON

Samuel Case(1)
Deborah Evans(2)
Gregory Tibbets(2)
Sarah Case(1)
David Miller(1)

ABSTRACT

Because very little nutritional research has been conducted on participants in ultra marathons and none has been reported on participants in cold weather ultramarathons, this study was undertaken with the purpose of examining the nutritional intakes and urine characteristics of participants in a cold weather ultramarathon. Fourteen participants (13 males and 1 female) in the IditaSport human powered ultramarathon volunteered to be subjects in this study. Pre and post race weights, skinfolds, and urine samples were taken. In addition, a dietary recall was done for the day(s) of the race. Data analysis revealed that a mean of 30,864 (17,143) kJ of energy were consumed with 71% of the energy coming from carbohydrate sources. Despite no significant change in body fat as measured by skinfolds, there was a significant ($P < 0.5$) decrease in body weight. Ketonuria and proteinuria were present in 83 percent and 92 percent of the subjects. When compared with participants in endurance events taking place in warmer climates, competitors in the IditaSport races consumed more energy with carbohydrates making up a greater percentage of their diets.

INTRODUCTION

The IditaSport Human Powered Ultra-marathon is a series of races in which participants choose one event from among the following advertised options:

1. a 120 km running race
2. a 120 km snowshoe race
3. a 250 km cross country ski race
4. a 250 km bicycle race
5. a 250 km triathlon made up of bicycling, skiing, and snowshoeing

The race begins in late February in Big Lake, Alaska and proceeds through the countryside to Skwentna. At Skwentna, the 120 km races (snowshoeing and running) end. For the 250 km races, Skwentna is the turnaround; from here the contestants return to Big Lake. Due to terrain features such as flooded streams, frozen streams, etc. that vary from year to year, the length of the course is not precise. For the most part, the course is fairly flat with much of the course proceeding on frozen rivers. The trail is packed prior to the competition by snow machines and is marked with lath strips that are painted with luminescent paint. Most of the contestants travel constantly with brief rest stops at the check points that are located approximately every 15 to 20 miles. Previous research has shown that ultra-marathoners and triathletes in races conducted in warm climates met or exceeded the Recommended Dietary Allowances (RDA) for all nutrients (20, 27). This study was undertaken with the purpose of determining the dietary intakes of participants in cold weather ultra-marathon events.

METHODS

The protocol for this study was approved by the Western Maryland College Institutional Review Board for Human Subjects Research. At a mandatory informational meeting two days prior to the start of the race, the research design was described to the race participants. From this group, 14 (13 males and 1 female) of 49 entrants, volunteered to be subjects and signed informed consent forms. The subjects included 2 of 4 entered runners, 1 of 9 entered snowshoers, 2 of 7 entered skiers, 1 of 2 entered triathletes, and 8 of 27 entered bicyclists.

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(1) Human Performance Laboratory, Western Maryland College, Westminster, MD 21157
(2) Dimond Athletic Club, Anchorage, Alaska

Correspondence should be addressed to:
Samuel Case, Ph.D.
Human Performance Laboratory
Western Maryland College
Westminster, MD 21157

Included as subjects were the second and third place runners, the second place snowshoer, the first and third place skiers, the first place triathlete, and the first, second, and third place bicyclists. The subjects' descriptive data appear in Table 1.

Following the meeting, each subject was weighed and skinfold measurements were taken at the chest, abdomen, and thigh sies on the males and at the triceps, suprailium, and thigh sites on the female. The skinfold data were used in the Jackson and Pollock generalized equations to calculate body density (8,9). Percent body fat was determined from the Siri equation (21). In addition, a urine sample was obtained to determine the presence of ketones, protein, or blood using Ames™ Labstix®. All measurements were repeated immediately upon completion of the race. At this time all subjects were asked to recall their dietary intakes from the morning of the race to the conclusion of the race. The dietary intakes were analyzed on a Macintosh Computer using the Mosby Diet Simple Analysis Program (13). If foods consumed were not in the data bank of the computer program, the nutrient content of the foods was obtained from food labels or by calling the manufacturer. The amount of these nutrients was then added to the totals obtained by the computer program. Since the race may have taken the participants either a greater or lesser amount of time than a 24 hour period (mean time for completion = 24.5 hrs.), the amount of the nutrients consumed was corrected to a time period of 24 hours for purposes of comparison to the RDA. Statistical comparisons of the pre-race and post-race data were accomplished using matched t tests, while comparisons between participants in the different races were accomplished using analysis of variance. Because of the small number of subjects, skiers and triathletes and runners and snowshoers were grouped together for this analysis.

RESULTS AND DISCUSSION

There were no differences in any of the variables between the participants in the five races. As can be seen in Table 2, the energy intake on race day ranged from 13846 kJ to 75138 kJ with a mean of 30864 kJ. The energy intake in the present study is much higher than the 4200 to 16800 kJ. and 11951 kJ reported in the studies of Newmark et al., who studied ultramarathon runners (14) and Worme et al. (27) who studied triathletes (swim, bike, run). Much of this difference can be accounted for by the fact that the races in the present study took longer to complete, involved more strenuous activities, and took place in colder weather than the cited studies. The energy costs for a 70 kg person for the activities comprising IditaSport have been estimated as follows: biking @ 10 mph, 32 kJ/min.; running @ 8.5 min/mile, 58 kJ/min; skiing, between 43 and 82 kJ/min, depending on grade; and snowshoeing, 63 kJ/min. (5, 11). The fact that the races took place on snow covered terrain also added to the energy cost. Smolander discovered that walking in soft snow required 3 times as much energy as an identical trial on a treadmill (23). Furthermore, the exercise heart rates were about 50 beats/per min. higher during the snow trial. During this particular IditaSport, since most of the snow was pre-packed by snow machines the elevation in the energy cost of running was probably not of the magnitude indicated in the study of Smolander (23). However, the energy cost for biking was probably considerably greater than the value listed above. Even though the snow was prepacked, the tires of the bikes sank in because of their relatively small surface area. In addition, many bikers on occasion had to dismount and push their bikes along the trail.

As far as the added demands of the cold are concerned, Shephard has documented very well the increased energy demands that cold weather activity places on the body (18). He cited as causes increased carbohydrate and fat metabolism that are mediated by increased circulating catecholamines, altered sensitivity to catecholamine receptors, and decreased amounts of circulating insulin. Further, he mentioned decreased mechanical efficiency of muscle contraction in the cold. Using the above information to conservatively estimate the energy cost of participation in the IditaSport events can be done by multiplying the mean time for completion of the events (24.5 hrs.) by the energy cost of each event. This yields values of 47,040 kJ, 92,610 kJ, and 85,620 kJ for biking, skiing and snowshoeing, and running respectively. Adding an allowance for the snow and the cold might conservatively raise these estimates

Table 1.

Physical Characteristics of IditaSport Participants Mean ± s.d.

Age (yrs.)	Pre-race Weight (kg.)	Post-race Weight (kg.)	Pre-race % Fat	Post-race % Fat
33.9 (7.8)	70.1 (6.9)	68.8 (5.9)*	9.46 (4.1)	9.0 (3.6)

* $P < 0.05$

by 10 to 50%. At any rate, the amount of energy expended during the race was probably greater than the mean amount of energy consumed during the race. The average weight loss by the participants was 1.3 kg ($p<0.05$) (See Table 1). It is difficult to draw a conclusion as to whether or not this was a result of fat loss or water loss. Although skinfold measurement revealed a slight, but statistically insignificant, fat loss of 0.46 percent, previous research has shown that skinfolds are affected by state of hydration, activity, and blood flow changes (7). The fact that the weight loss was so small for races of this duration is noteworthy, and is due to the educational efforts of the race organizers during the pre-race meeting. At this meeting the dangers of dehydration in a cold environment were emphasized in both oral presentations and in written handouts.

The analysis of the diets in terms of energy nutrients revealed that the participants were consuming a carbohydrate rich diet (See Table 2). The carbohydrate content was well above the 55 percent recommended for the American population (25), but was in keeping with the 60 percent to 70 percent recommended for endurance athletes (19, 22). The protein content was a bit below the 15 percent that is recommended by most authorities (5, 25). However, this is probably not of any great consequence since protein sources play a relatively minor role in contributing to energy production during activity (5, 22). The fat content of the diet on race day (18 percent) was lower than the recommended level of 30 percent of the day's energy intake (5, 25). However, many authorities feel a diet that is 10 percent fat provides sufficient quantities of the essential fatty acids (26). It is important to note that for cold weather endurance activities, fat metabolism plays a more prominent role than for activities conducted in warmer weather. Therefore, it is essential that fats not be totally excluded from the diet in favor of carbohydrates (18). Obviously, good nutrition does not require minimum amounts of each of the nutrients each day; rather, good nutrition is a matter of getting the proper amounts of nutrients over a period of time.

As far as the RDA were concerned, despite large variations in intake, the mean vitamin and mineral components of the athletes' diets were in excess of the RDA needed for the respective age groups (2). This has been documented in other studies (20, 27), and is due to the fact that athletes in these types of activities consume prodigious amounts of food during the contests; this more than makes up for any individual nutrient deficiencies that may be present. As with the recommended dietary percentages of energy nutrients, obtaining the RDA for each and every nutrient on a daily basis is not very important. As a matter of fact, the RDA were designed for a sedentary population, and standards have not yet been devised for different levels of activity (27). For this study, they only served as standards for comparison.

Two nutrients that can have deleterious effects during performance if they are not present in sufficient amounts are the electrolytes sodium and potassium. These two electrolytes are very important for proper neuro-muscular function, including preventing muscle cramps in endurance events. Noakes et al. have shown that there is not much evidence of hyponatremia in endurance athletes competing in the Comrades Marathon, but the importance of proper electrolyte balance should not be minimized (15). In their study only 0.3 percent of the participants suffered from hyponatremia; but in those who collapsed, the frequency of this disorder rose to 9 percent.

The most frequently consumed foods by the competitors during the race were high carbohydrate energy foods that are marketed in both solid and liquid forms. Among these were Power Bars, Ultra Fuel Bars, Cliff Bars, Fibars, Gatorlode, Exceed, and Metabolol. In addition, Carnation Breakfast Bars and Pop Tarts were eaten by some contestants. One contestant ate 6 Power Bars, 4 Cliff Bars, 3 Ultra Fuel Bars and 16 quarts of Gatorlode and Ultra Fuel during the race. This was in addition to the meal of spaghetti and/or pancakes that was provided at the turn around checkpoint in Skwentna!

Several of the athletes (21 percent) complained of various forms of intestinal distress including gas, cramps, and diarrhea. This phenomenon has been documented in other research. Worobetz and Gerrard (28) noted that 81

TABLE 2.

Energy Nutrient Content of IditaSport Participants' Diets Mean ± s.d.

	ENERGY (kJ)	CHO %	PROTEIN %	FAT %
ALL	30864 (17143)	71.6 (14.6)	9.6 (3.5)	18.7 (13.3)
RUN & SS[1]	25835 (3363)	63 (17.5)	10.0 (2.7)	27.0 (15.0)
BIKE	35405 (21830)	74.1 (16.0)	9.4 (3.4)	16.5 (15.1)
SKI & TRI[1]	23809 (6015)	74 (3.6)	10 (6.0)	16 (9.2)

[1] Because of small sample sizes runners and snowshoers, and skiers and triathletes have been grouped together.

percent of their subjects reported some form of gastrointestinal distress while running. Sullivan (24) and Worme et al. (27) observed the same symptoms in 25 percent and 50 percent of their subjects respectively. The causes of these problems have been attributed to everything from the demands of the exercise to a sudden change to unfamiliar sources of energy when high carbohydrate ergogenic aids are consumed (17). Newmark et al. have speculated that candy bars may contribute to gastrointestinal distress because the relatively hyperosmolar concentration has increased fluid requirements for digestion (14). In the present study 13 of the 14 subjects used some form of high carbohydrate ergogenic aid. The incidence of distress during this competition is similar to that found by Sullivan (24). To prevent these problems during competition, authorities on exercise recommend consuming the foods that will be eaten in competition during training sessions (17).

The incidence of ketones, blood, and protein in the post race urine was 83 percent, 17 percent, and 92 percent respectively. These phenomena have been documented in other studies of exercise and kidney function (3, 4, 10, 16). Ketonuria has been attributed to a lack of carbohydrate availability and the resulting shift to fat metabolism (3,10). In the present study, as pointed out earlier, the energy intake was probably not equal to the energy output. Hematuria and proteinuria have been observed for many years and have been attributed to tissue damage resulting from the exercise and to an alteration in the transport mechanism in the kidney (6, 16). Since these analyses were accomplished using dipsticks, there is the possibility that the positive reactions for blood were caused by the presence of myoglobin. In fact, Bank has noted this phenomenon following carbohydrate loading - a practice followed by many of the competitors (1). Unfortunately, the urine was not examined microscopically so that the actual cause could be pinpointed. Mittleman and Zambraski have suggested that the proteinuria is mediated by prostaglandins (12).

In summary, participants in the Iditasport race, despite losing weight, consumed greater quantities of energy than those competing in endurance competitions conducted in warmer climates. Furthermore, a larger percentage of their diet was derived from carbohydrates. Most also used nutritional supplements to increase their performance, and this might be the cause of the gastrointestinal distress seen in many of the competitors. In addition, most participants exhibited proteinuria and ketonuria as a result of the strenuous muscle activity. Competitors in these types of cold weather activities should be cautioned to remain well hydrated and to consume adequate amounts of energy in the form of carbohydrate and to a lesser extent fat. Furthermore, if they are going to consume high energy food supplements during the competition, they should practice eating these foods during training sessions to obviate gastrointestinal distress.

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Benzene in Alaska

Mary Ellen Gordian, M.D., M.P.H. (1)
Gerald Guay (2)

ABSTRACT

Alaska Department of Environmental Conservation took indoor and ambient air samples during the winter of 1992-93 in Fairbanks. The samples showed a significant increase in the level of benzene in the air between December and February. The highest levels occurred in indoor air and exceeded workplace standards in garages. These findings were corroborated by NIOSH and OSHA personal monitoring studies done at the same time in Fairbanks and by blood samples taken by Centers for Disease Control and Prevention from workers exposed to gasoline in December and February. The blood samples showed a 300 percent increase in the amount of benzene in worker's blood after MTBE was taken out of gasoline. The most likely cause of this air pollution is gasoline evaporation and exhaust emissions. The benzene content of Alaska gasoline is 5 percent which is the highest in the nation. In view of these findings the Environmental Protection Agency is funding a new study of indoor air organic compounds including benzene in Anchorage, Alaska.

HISTORY AND INTRODUCTION

In the winter of 1992-93 the city of Fairbanks and the Municipality of Anchorage used methyl tertiary butyl ether (MTBE) as an oxygenate in the gasoline sold in those cities. This was done to comply with the Clean Air Act of 1990 to reduce the carbon monoxide in automobile exhaust. There was an immediate 14 cents per gallon price increase and the odor of gasoline was altered by the additive. Citizens began to complain of headaches, respiratory symptoms, rashes, dizziness, and other symptoms which they associated with the introduction of the oxygenate.

In December of 1992 the Alaska Department of Environmental Conservation (ADEC) began taking air samples in Fairbanks, Alaska, and testing them for MTBE and a large number of volatile organic compounds. Sampling began the first week of December. ADEC took outdoor air samples and samples from inside garages and inside city vehicles. At the same time that these samples were being taken investigators from the Center for Disease Control and Prevention (CDC) were taking blood samples looking for MTBE as well as other volatile organic compounds. Because most people spend the majority of their time indoors in Fairbanks in December, ADEC did indoor air sampling as well.

On December 15, 1992, the Governor suspended the oxygenated fuel program in Fairbanks because of citizen complaints. In February, ADEC returned to Fairbanks to repeat the air samples both indoors and out. The purpose of the air sampling was to survey the environment for MTBE and volatile organic compounds. The sampling was done in response to citizen complaints to determine what levels of MTBE were in the air. Both ambient outdoor air and the air in specific environments such as workplace, school, post office, and one residence were sampled.CDC also returned to Fairbanks to repeat the blood sampling for MTBE and other components of gasoline. This was a pilot project as no one could predict what might be found. The complaints in Fairbanks had never occurred elsewhere. Because there was evidence from tailpipe emissions studies that MTBE increased the concentration of formaldehyde in auto exhaust emissions, there was concern that formaldehyde might be accumulating in the arctic environment and causing symptoms. Formaldehyde and other carbonyls were also included in the testing.

The results of the air sampling showed that MTBE was present in the environment as well as other volatile organic compounds. However, a closer inspection of the data showed that benzene, a known carcinogen, appeared to increase after MTBE was discontinued. Even though the number of samples taken was small, a consistent significant pattern of increased benzene concentration was seen in indoor air samples, and the same pattern although not statistically significant was seen in the ambient air samples. The finding of increased benzene concentrations in indoor air is a significant public health concern. Although personal exposure monitoring was not done, the blood samples from workers showed an increase of 300 percent in blood benzene indicating a very significant increase in exposure.

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(1) Medical Officer, Department of Health and Human Services, Municipality of Anchorage, P.O. Box 196650, Anchorage, Alaska 99519-6650.

(2) Project Manager, Ambient Analysis Group, Division of Environmental Quality, Alaska Department of Environmental Conservation, Juneau, Alaska.

METHODS

Sampling for hydrocarbons was performed using a six liter, passive stainless steel canister sampling system. Each monitoring system employed a mechanical flow controller to establish an eight-hour sampling period. Certified "clean" canisters were exposed to sample air and sent to the Oregon Graduate Institute in Beaverton, Oregon (December) and Environmental Protection Agency's (EPA) Atmosphereic Research and Exposure Assessment Laboratory (AREAL) in Research Triangle Park, North Carolina (February) for analysis. Both laboratories use EPA's compendium Method TO-14 for the analysis of benzene. Airborne concentrations of formaldehyde and acetaldehyde were evaluated with portable aldehyde samplers which pulled sample air through two silica gel impregnated, 2, 4-dinitrophenylhydrazine (DNHP) sampling cartridges for two, 4-hour periods. This sampling schedule was revised to one sampling cartridge every eight hours once it was shown that measured concentrations were not saturating the cartridge. DNHP sample analysis was performed through the Desert Research Institute in Reno, Nevada, (December) and AREAL (February). Both laboratories used EPA's compendium Method TO-11 for analysis of aldehydes.

Indoor and vehicle sampling times and locations were selected by CDC and designed to meet worker shift schedules. Ambient monitoring was designed around zones of maximum public exposure and established carbon monoxide sampling sites. Ambient samples were taken around the CDC schedule and were staggered to allow the technician to service them following the eight hour monitoring schedule. In addition to air samples, the field technicians recorded sample air temperature and atmospheric pressure for flow rate correction. General weather observations were recorded for all ambient samples to include outside air temperature. Quality assurance was done on random samples. EPA re-analyzed some of the samples done in the Oregon Lab for quality assurance.

ANALYSIS

The data were entered and analyzed using EPI-INFO, statistical software. The samples taken in December 1992 were aggregated and were thought to be representative of the quality of the air while MTBE was in use. This was substantiated by the relatively high value of MTBE found in the samples.

The samples taken in February and the first week of March were aggregated as representative of air quality after MTBE was discontinued. This was substantiated by a drop in amount of MTBE in the air, although it is important to note that it never disappeared completely. Means were calculated for repeat samples from the same

Table 1. **Air Sampling in Fairbanks**
Indoor Air

Chemical	Month	N*	ppb Mean	S.D.	Range	T-test
Garages						
Benzene	December	6	94.02	±65.35	18.5 - 175.3	p = 0.03
	February	11	191.62	±88.56	83 - 350.94	
MTBE	December	6	304.67	±284.01	54 - 811	p = 0.01
	February	11	59.77	±51.27	5.9 - 141	
Formaldehyde	December	11	17.20	±9.01	4.4 - 35.2	p = 0.0001
	February	11	34.61	±6.83	28.5 - 51.8	
Non-Garage						
Benzene	December	10	5.89	±3.61	1.29 - 12.54	p = 0.01
	February	13	20.37	±17.71	2.63 - 51.03	
MTBE	December	10	6.58	±4.54	1.46 - 16.73	p = 0.21
	February	13	7.36	±10.21	0.0 - 30.33	
Formaldehyde	December	10	12.75	±8.73	2.75 - 29.70	p = 0.05
	February	13	20.22	±8.79	3.87 - 32.24	
Vehicles						
Benzene	December	7	28.63	±36.31	7.6 - 109	p = 0.76
	February	6	34.28	±28.94	5.1 - 79.2	
MTBE	December	7	83.57	±113.72	12 - 335	p = 0.015
	February	6	11.36	±12.87	0.4 - 35.8	
Formaldehyde	December	7	3.43	±2.67	0.78 - 7.86	p = 0.03
	February	6	7.87	±3.91	2.23 - 13.94	

Table 2. Air Sampling in Fairbanks
Ambient Air

Chemical	Month	N*	ppb Mean	S.D.	Significance
Major Intersection					
Benzene	December	7	6.50	±3.99	p = 0.125
	February	6	10.65	±5.11	
MTBE	December	6	4.55	±3.74	p = 0.006
	February	6	1.12	±0.57	
Formaldehyde	December	6	2.40	±1.67	p = 0.03
	February	5	4.99	±1.78	
Elementary School					
Benzene	December	6	5.68	±5.15	p = 0.68
	February	6	4.71	±2.46	
MTBE	December	6	4.69	±4.50	p = 0.004
	February	6	0.53	±0.35	
Formaldehyde	December	5	5.55	±4.06	p = 0.62
	February	6	4.49	±2.72	
Post Office					
Benzene	December	8	11.79	±9.38	p = 0.76
	February	5	13.42	±8.60	
MTBE	December	8	8.16	±3.72	p = 0.005
	February	5	1.85	±1.52	
Formaldehyde	December	8	4.69	±3.54	p = 0.0007
	February	5	14.36	±3.33	
Private Residence Outdoors					
Benzene	December	6	7.48	±6.04	p = 0.20
	February	5	12.07	±4.76	
MTBE	December	6	7.38	±7.85	p = 0.03
	February	5	1.00	±1.06	
Formaldehyde	December	4	4.64	±4.62	p = 0.39
	February	4	5.54	±1.20	
Service Station Fuel Island					
Benzene	December	1	18.68	—	—
	February	4	27.74	±6.32	
MTBE	December	1	29.88	—	—
	February	4	5.47	±5.59	
Formaldehyde	December	1	8.78	—	—
	February	3	5.78	±2.96	

location and December was compared to February. Samples from garages and the ambient air samples were aggregated and compared. Three other sites were sampled including one residence, one school and the post office. These were compared individually and aggregated to represent non-garage indoor air. The results are presented in Table 1, ambient (outdoor) air, and Table 2, indoor air.

RESULTS

The results show an increase in the amount of benzene and formaldehyde in indoor air *after* MTBE reformulated fuel was discontinued.

Ambient Air - The amount of benzene in ambient air in Fairbanks generally increased, but not significantly, after MTBE was discontinued. MTBE did decrease significantly in ambient air between December and February. The amount of formaldehyde in ambient air increased significantly at two sites *after* MTBE was discontinued.

Indoor Air - Indoor air benzene concentration increased significantly after MTBE was discontinued in garages and non-garages, but not in vehicles. Vehicle data was difficult to interpret because different vehicles were tested in December than in February and the variation between vehicles is great. Although more garages were tested in February, the garages that were tested in December were also retested in February. In

December out of 6 samples taken only two exceeded the National Institute for Occupational Safety and Health (NIOSH) recommended exposure limit (REL) of 0.1 part per million (ppm) for benzene. In February out of eleven samples taken, ten samples exceeded the NIOSH REL standard. The mean concentration of benzene had doubled, while the concentration of MTBE had dropped significantly. In the samples taken at the school, post office, and residence benzene increased from a mean of 5.89 parts per billion (ppb) in December to a mean of 20.22 ppb in February which was significant. The concentration of MTBE in these non-garages was low even in December and was not significantly reduced. Formaldehyde increased significantly *after* MTBE was discontinued which was unexpected.

The concentration of benzene was positively correlated with the concentration of MTBE in both indoor and outdoor samples both in December and February. That is, in locations where benzene was high, MTBE was also high, even though the ratio between benzene and MTBE changed from December to February.

DISCUSSION

This study was not vigorous enough to assess whether the increase in benzene which was seen in February resulted from the discontinuation of the use of MTBE or from other factors. However, the results of this study are consistent with studies from Finland which showed that the lower the temperature the higher the absolute emission reductions of hydrocarbons that can be achieved by using gasoline oxygenated with MTBE. The Finnish study showed that the tailpipe emission of volatile hydrocarbons including benzene from vehicles using gasoline oxygenated with MTBE is reduced by 10 percent at 20 degrees Celsius and 20 percent at -20 degrees Celsius .

Because of the focus on fuel additives, we began to look at the content of the fuel. Gasoline in Alaska has a benzene concentration at 5 percent which is the highest in the country. The average concentration is 1.5 percent benzene. Alaska gasoline has a Reid vapor pressure (RVP) of 15 psi which indicates high volatility.

Health Risks — None of the benzene concentrations found in Fairbanks except that which was found in garages exceeded the NIOSH recommended exposure levels for workplaces. NIOSH investigated eleven facilities some of which were the same as were studied by ADEC in Fairbanks from February 9-19, 1993. They also found benzene concentrations ranging from 0.02 to 0.57 ppm, which exceeds the OSHA action level. State Occupational Safety and Health Agency (OSHA) also had found even higher levels in December when they did personal exposure studies on workers in automobile repair shops. Results of personal monitoring in December showed that workers were exposed to over 1 ppm which is the OSHA permissible exposure limit (PEL). OSHA did look primarily at workers that they expected to have had high exposures because of their high risk work. CDC confirmed this high exposure with blood tests that showed concentrations from 0.42 ppb to 1.3 ppb, 300 percent increase in blood benzene after MTBE was removed. It is unknown how representative the single residence tested by ADEC for indoor air benzene was. The single residence showed a 400 percent increase in benzene from 5 ppb in December to 20 ppb in February. Subsequent indoor samples from Anchorage without MTBE in gasoline showed an average indoor concentration of benzene to be 19 ppb. (Work in Progress) Whether these indoor levels may affect women and small children who spend the majority of their time in the home is unknown at this time.

These concentrations may have long-term health consequences as both ambient air and indoor residential air were higher than has been found in other major industrial cities. There is no industrial source of benzene pollution in Fairbanks. The strong positive correlation of benzene with MTBE throughout the study indicates that both have gasoline as a common source. Alaska fuel is the most common source of benzene in the environment.

Health Effects — Benzene is a known human carcinogen. It has been shown to cause bone marrow toxicity and lymphohematopoietic cancer. The National Institute for Occupational Safety and Health recommends a time weighted average (TWA) of no more than 0.1 ppm for 10 hours. Sufficient evidence regarding its toxicity has been collected to allow the American Conference of Governmental Industrial Hygienists (ACGIH) to lower the occupational exposure known as threshold limit value (TLV) to 0.1 part per million for an 8 hour day. There is no threshold limit set for continuous exposure such as from ambient air or indoor residential air. This area is under investigation at the present time. The Environmental Protection Agency estimates that a lifetime exposure to 0.004 ppm (4 ppb) benzene in air would result in one additional case of leukemia for every 10,000 people exposed. The concentrations that were found in Fairbanks in ambient air as well as indoor air were higher than 4 ppb in December, and increased to more than twice that level outdoors, and four times that level indoors in February.

Alcohol and phenobarbital ingestion increase the rate at which the toxic metabolites of benzene are formed. People with rapidly synthesizing bone marrow are at greater risk. Experimental studies show that intermittent exposure to low levels of benzene cause chromosomal aberrations. Benzene levels at gas stations during refueling have been measured as high as 6.6 ppm in the lower 48. Gas station exposure studies have not

(continued on page 36)

Memo

To: **President Clinton**
Members of the House and Senate

From: **The 300,000 member physicians of the American Medical Association**

Re: **Getting the job done on health care**

We believe there are several workable measures that can be passed now to help achieve our ultimate goal – making quality health care affordable and accessible to all Americans.

As the country awaits the State of the Union address, the member physicians of the American Medical Association set forth these practical recommendations to improve health care for our patients.

Insurance Reform – Pass insurance reforms that will make sure Americans will not lose their coverage if they change jobs or get sick.

Medicare Reform – Reform our Medicare system so it will be there for the next generation of elderly and disabled.

Medical Savings Accounts – Make MSAs available so people can pay for routine medical care with pre-tax dollars.

Patient Protections – Enhance patient choice, disclosure and assure greater physician involvement in corporate decisions about patient care.

Liability Reform – Enact meaningful liability reform to ensure fair compensation to patients with legitimate claims while eliminating excessive malpractice awards that lead to defensive medicine.

Regulatory Relief – Free both physicians and patients from the ever-increasing burden of needless and wasteful paperwork, regulations, obsolete anti-trust rules and red tape.

Medical Education and Research – Protect medical education and research so that we can find cures for killers like AIDS and cancer.

Public Health Problems – Fight social problems like violence and smoking — problems that cost billions of dollars and millions of lives.

These measures are sensible things we can do now that will make a difference for all of us. So, as we begin our nation's 104th Congress, we renew our pledge to the health of America. As the voice of the medical profession, we pledge to do everything we can to help make these things happen. It is our contract with America, and we fervently hope that every American will join us.

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Yohimbine exerts a stimulating action on the mood and may increase anxiety. Such actions have not been adequately studied or related to dosage although they appear to require high doses of the drug. Yohimbine has a mild anti-diuretic action, probably via stimulation of hypothalmic centers and release of posterior pituitary hormone.

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Indications: Yocon® is indicated as a sympathicolytic and mydriatric. It may have activity as an aphrodisiac.

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Warning: Generally, this drug is not proposed for use in females and certainly must not be used during pregnancy. Neither is this drug proposed for use in pediatric, geriatric or cardio-renal patients with gastric or duodenal ulcer history. Nor should it be used in conjunction with mood-modifying drugs such as antidepressants, or in psychiatric patients in general.

Adverse Reactions: Yohimbine readily penetrates the (CNS) and produces a complex pattern of responses in lower doses than required to produce peripheral a-adrenergic blockade. These include, anti-diuresis, a general picture of central excitation including elevation of blood pressure and heart rate, increased motor activity, irritability and tremor. Sweating, nausea and vomiting are common after parenteral administration of the drug.[1,2] Also dizziness, headache, skin flushing reported when used orally.[1,3]

Dosage and Administration: Experimental dosage reported in treatment of erectile impotence.[1,3,4] 1 tablet (5.4 mg) 3 times a day, to adult males taken orally. Occasional side effects reported with this dosage are nausea, dizziness or nervousness. In the event of side effects dosage to be reduced to ½ tablet 3 times a day, followed by gradual increases to 1 tablet 3 times a day. Reported therapy not more than 10 weeks.[3]

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Sexually Speaking . . .

Mary Cavalier, M.S.

The Power of Thought

I have often referred to the use of cognitive therapy in treating sexual dysfunction. I thought I would illustrate one approach which can be helpful: Positive Affirmations. Unfortunately, positive affirmations often conjure up images of the '60s or "new age" and in turn, people may shy away from using them. But if one looks at the approach objectively, one may see, if not experience, the benefits.

On a daily basis, we are consistently giving ourselves messages. Simple, yet powerful messages. "That was a stupid thing to do", "Why don't you think first", "You'll never get anything right", "No matter what I do it's not good enough", etc. The overall purpose is to stop spontaneous action which puts us at risk of trying something new or different. The "I can'ts" control us.

Sexually speaking, simple messages such as "bad girl/boy", "shame on you", "tramp", "whore", "it feels too good", "you're not good enough", are powerful enough to dampen sexual desire and inhibit orgasm. These messages are often old and perhaps not even conscious. Yet as with the messages mentioned above, they can rule one's life.

So what to do? It does not work to attempt to tell oneself not to think that way. The mind likes to fill in empty space and usually fills it with what is familiar; e.g., more negative messages. So one needs to create positive messages to replace the negative ones. Not so easy. I liken it to learning a foreign language. One needs to practice, practice, practice and it will feel very uncomfortable at first. I often prescribe a phrase (one the patient has created themselves) to be repeated at least 30 times a day. Highlighted are some examples which you can cut out and give to patients. As with any treatment, secondary gains play a major role in accepting changes. If the patient reacts strongly to some of these statements, there may be underlying issues which need to be addressed.

"It is safe to feel sensual feelings."

"I enjoy being sexual."

"I am a sensual being."

"I enjoy being touched."

"It is safe to feel good."

"It is OK to be vulnerable with my partner."

My Turn - Primary Care Provider Important to Children

Kenneth Moss, M.D.[1]

Every child should have a central place where medical care is received. This is true for all people, but it is especially true for a child, since he or she is in the beginning of life and is a growing, changing, dynamic and developing being from birth until independent adulthood.

"Health-care reform" is an expression constantly in the news, promising change with all sorts of schemes, organizations, plans and services. A parent can get confused with the offerings. However, she or he should choose a provider for primary care, and see that most of the care is given or centralized there.

This "medical home" should include a comprehensive record of not only encounters for illnesses and crisis situations, but also of regular growth, development and immunization status.

The parent, child and provider should all feel comfortable with one another. The doctor or clinic and staff should develop a feeling of mutual trust and confidence with the individual.

The best place for a medical home for your child is with a personal physician, a family practitioner or pediatrician (or a family practice or pediatric clinic). The American Academy of Family Practice and the American Academy of Pediatrics support this concept.

There are two good reasons for this centralized care. First, it provides a comprehensive record of health maintenance and problems from birth on. Second, it allows the young person to develop a relationship with a care-giver over time. If the family moves to a different location, a new medical home can be found.

The physical medical record itself belongs to the doctor, clinic or hospital providing care, but the information belongs as well to the child and family. This information is available and accessible, and is meant to assist in health maintenance and management throughout life.

Most humans, and perhaps especially most Americans, have a desire to be kind, helpful and generous to children. This has generated many programs, plans, camps, clinics, campaigns, centers, etc., for children. This has been good for children's health, but sometimes contributes to patchwork or non-continuous care.

In this community, for instance a child may receive care from a private physician, the hospital emergency room, the Urgent Care Center, Juneau Public Health Center, SEARHC clinic, Coast Guard clinic, visiting specialty clinics, school nurse, teen clinic, Shriner's Clinics and Hospitals, Muscular Dystrophy Association, etc., to name only a few.

I have had children receive immunizations from as many as five sources. This is not to say that multiple encounters are bad or inferior. These sources may be excellent and extremely necessary. The point is, some centralization of these records in a medical home of a primary care provider, the family physician or pediatrician clinic, is to the advantage of the child.

To make an analogy: If you buy a fine new car, you get a maintenance schedule of checkups to keep it working best and to age well without undue wear. The child deserves at least that much.

And what type of health maintenance and checkups are advisable for the infant and child? During the first year, several. A complete exam at birth and at 10 days or so to begin with; then at two months, four months, six months, and possible at nine and 12 months.

These visits approximate the times of immunizations.

In the second year, a little less often — usually at 15 months, 18 and 24 months.

Checkups about once a year during the preschool years are advised, and every one to two years during the school years.

Illnesses, injuries, and problems should be addressed at any time they occur.

And what should you expect from your primary health-care provider? An assessment and evaluation of normal growth and development of the child, and deviations from it, as well as a source of care for illnesses when they occur.

Also expect guidelines for when to call for help for the child, and recommendations for home care; an explanation for illnesses or conditions when they occur, such as: What is the cause? Is it contagious? What to expect? How long will it last? What signs to look for if worsening?

When supplemental care, such as the emergency room, is given, it should be recorded in the child's medical home record. If specialty care is given, it should also be recorded.

The desire to do the best job of child-rearing and child-caring is universal. Parents are constantly seeking

(continued on page 36)

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(1) Juneau pediatrician. 1600 Glacier Avenue, Juneau, Alaska 99801.

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History of Medicine in Alaska

Paul Isaak, M.D.

Three major themes run in the life of Paul Isaak: homesteading, flying and medicine.

His father, William, came to South Dakota from Russia at the age of three. He homesteaded in Parkston where Paul was born on July 10, 1921.

He went to school through the eighth grade, but while he wanted to go on to high school, his father decided that he was needed to work on the homestead.

When World War II broke out, Paul Isaak was declared 1-A which meant being drafted. He wished to become a pilot but was denied by the Air Force because he had not been to high school. He enlisted in the Marine Corps as a private. He saw much action in New Caledonia, Guadalcanal, the Marshall Islands, the Mariana Islands and Okinawa. At the end of the war, he was sent to Tientsin, China to take guns from the Japanese who had occupied that country since the thirties.

On Okinawa, he recalls a Japanese air raid in which four or five planes landed on the airstrip. The pilots set fire to the parked U.S. airplanes before being shot.

> His firm philopophy is that one should set goals and then apply himself to attain them.

Returning home after three years overseas, he worked for his brother as a welder for one and a half years — a useful skill to be exploited later.

At the suggestion of his VA adviser, he took the GED exam and passed. But this did not entirely satisfy him, so he enrolled as a very mature high school student at Augustana Academy in Minnesota. After he completed the first semester, the principal told him that if he applied himself for the second semester, he might get a diploma. He took sixteen subjects and was graduated after seven months.

He went on to Augustana College for four years. He worked summers for a cement contractor at Sioux Falls, South Dakota. His advisor was a chemistry professor so he majored in chemistry. He entrolled at the University of South Dakota School of Medicine, which at the time was a two-year school, and received his MD degree at the University of Nebraska in 1956, serving a rotating internship at Swedish Hospital in Seattle.

He met Amy Christensen during his freshman year at college. She was a senior and was secretary to the college president. They were married in 1948 and by the time Paul finished college, they had two children. When he had completed medical school, the Isaaks had four children. Once they arrived in Seward, Alaska, a fifth child was born. All three daughters and two sons currently live in Alaska.

Since Alaska had always interested him he served a three-month tour as a physician with Alaska Packers cannery in Bristol Bay. While he was stationed at Ugashik he got to know everyone in the village and he covered the territory from Pilot Point to Ugashik. He has fond memories of the Natives who were wonderful people. To get to King Salmon, he was flown in a tripacer by Orin Seybert who now operates Peninsula Airways.

This experience convinced him. The money he earned provided most of the capital for the move, but he also obtained a loan from the chief of surgery at the Mason Clinic. This loan was greatly appreciated since its only collateral, was future goodwill. Back in Seattle the Isaaks bought a trailer and hauled it up the Alaska Highway in its primitive days, accompanied by four small children.

In Seward in 1957, Dr. Isaak joined Dr. Deisher who was building a new clinic in which he rented space. A third doctor in town was Dr. Hall who had some training in surgery but who left after a year. Dr. Isaak bought his equipment mostly as an act of good will and moved one and half blocks into his own office.

Dr. Isaak learned to fly in Seward and eventually he and a friend bought a J-3 cub. Later he bought his own plane, a Pacer. He began to fly to Seldovia and Soldotna. Eventually, he rented a two-bedroom house in Soldotna for office hours. There was an airstrip behind his office which, in the winter, was not plowed and since his plane had no skis, he landed and took off from the highway. He began to fly patients to the hospital in Seward.

Dr. Isaak tells of a young man who was working on a drilling rig when a grease gun dropped from a 90 foot tower, pierced his helmet and skull and penetrated his brain. He did not lose consciousness. His father put a bandage on his head and took him to the doctor. Paul Isaak was busy but finally got around to the patient, who after all this, was still talking. Dr. Isaak put him on an old x-ray table which was used also as an examining table to look at the wound which was about the size of a quarter. He immediately flew son and father to Seward. Fortunately, Perry Mead, the only neurosurgeon in the state, was on his boat in Resurrection Bay. Perry and Paul operated without anesthesia. Perry probed to explore the limits of the damaage. They decided to leave well enough alone. After closing the hole, the man spent three or four days in the hospital. These were the glorious days when the patient was grateful to the doctor for what he could do and there was no danager of a malpractice suit. The patient suffered only some visual field loss.

Dr. Isaak acquired a Cessna 180, perhaps his favorite aircraft. Into this he once loaded a man and his wife who was in labor. They flew from Soldotna to within five miles of Seward which was socked in. Returning to Soldotna, the husband got his car and they started back to Seward. They got within five miles again, but at this point the woman delivered.

But there were deaths too. Once he landed on the road at Cooper Landing to find a man dead from a probable heart attack. At Kasilof he landed on the road near the home of a badly burned child. Paul Isaak flew him to Anchorage where he was stablized and then flown to a burn center in Texas where he died.

When asked about the airplanes he has owned, Dr. Isaak admits to a J-3; a Pacer; two Cesna 180s, the second one new with a stretcher; a 1975 Supercub which he sold to Dr. Declan Nolan; a 1979 Supercub which he flew up from Outside plus several rebuilt aircraft. His first Supercub cost $14,000; the second four years later cost $25,000. Rebuilding airplanes is a hobby. Currently he has the frame of an airplane in a jig which is a heavy frame that maintains alignment. In rebuilding airplanes he has used the welding skill he acquired while working long ago for his brother. This interview was briefly interruped by a man looking for a propeller.

A new hospital opened in Soldotna in 1972, but that is another long story. Dr. Isaak worked for more than five years to achieve this. He had built a clinic with Dr. Gaede which was designed and built by Milt Echhaus of Los Angeles, an engineer and architect. Medical faciliies were his specialty and they were well satisified with the clinic. When Mr. Eckhaus was asked about designing and building a 30-bed hospital, he offered a "turnkey" job for the $400,000 SBA loan amount. The hospital board rejected him because they wanted an architect from Alaska. An Anchorage architect was hired, the project was started, but it soon ran out of money. The building sat for three years as an empty shell. Finally the borough took it over and completed it with an additional $1.5 million.

The Swanson River old field started up its exploration. Dr. Isaak was kept very busy medically with patients from the area. He worked with Dr. Gaede for over ten years.

At first Paul Isaak had practiced two days a week in Soldotna, flying in from Seward, then in 1960 he met a man from Kenai who knew people who had filed for homesteads but had not used the land. Dr. Wilcox in Anchorage was one. When Dr. Isaak acquired that homestead, he did use it, starting by building an airstrip. Meanwhile the Isaaks lived in a three-bedroom trailer parked behind the office. Eventually, he built a house and three hangers. The last, with help from his sons, is enclosed and heated.

Dr. Isaak is youthful looking and energetic. He did not keep count of the several thousand babies he delivered. He ran an air ambulance service when this was the most effective way to care for patients. He is now retired to his favorite hobby, airplanes, on his homestead. His firm philopophy is that one should set goals and then apply himself to attain them.

Gwynneth Gminder Wilson
Alaska Medical Alliance

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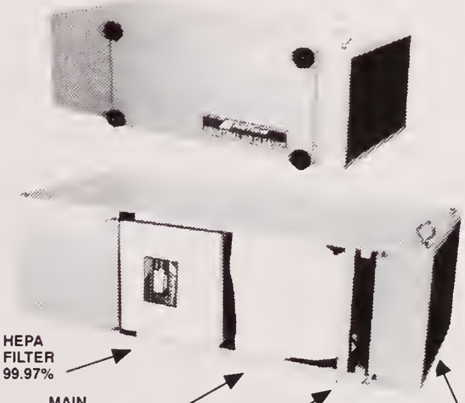

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(continued from page 28 - Benzene)

been done in Alaska.

Another significant source of benzene exposure is cigarette smoke. Indoor residential levels of benzene can be elevated if anyone is smoking. The smoking policy of the workplaces in this study is unknown. Indoor smoking is not a likely cause of benzene pollution in the school or the post office. There were no smokers in the residence that was studied.

This study raises many questions but leaves most of them unanswered. The Air Quality Section of Department of Health and Human Services at the Municipality of Anchorage and Alaska Department of Environmental Conservation have just completed a year long ambient air study of volatile organic compounds including benzene. The Municipality has received a grant from EPA to do a survey of indoor air volatile organic compounds including benzene which may answer some of these questions. Whether these levels are high enough to pose a threat to health is yet to be answered. Certainly workplace levels do exceed NIOSH recommendations and personal exposures for automobile repair workers are in the hazardous range. Efforts should be made to improve ventilation in garages. Reducing the benzene content of Alaska gasoline would protect not only workers, but may protect the general public as well.

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(continued from page 32 - My Turn)

and provided with information by the media and by relatives or others. Some of this information is excellent and accurate, and other times it may be confusing, conflicting or alarming. . .

Helping the parent choose information wisely is part of the primary care provider's duty.

The parent, of course, knows the child better than anyone else. The parent, child and physician and staff should be a team. The best teamwork is done through a principal care-giver.

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GLIMPSES OF ALASKAN MEDICAL HISTORY

Edited by Robert Fortuine, M.D.

A visit to the hot springs near Sitka (1842)

When Sir George Simpson (see previous article) returned to New Archangel in March of 1842, he had occasion to use the hot springs located some distance to the south of the capital. These springs had been known to the Russians since their earliest settlement of the area; Captain Yurii Lisianskii had described in some detail his visit there in 1805. The Tlingit, of course, had also known of them and had been enjoying their refreshing qualities for countless generations. By the time of Simpson's visit, the Russians and the Sitka Tlingit—not always on the best of terms in the best of times—had come to an understanding about sharing peaceably nature's healing waters. Not so peaceable, however, were the Stikine Tlingit, who some years later attacked the site, burned the cottages, and killed many of the Sitkan band enjoying the waters. The "hospital" was later rebuilt by the Russians and again used jointly. In the early decades of the twentieth century, the springs were the site of a fashionable "spa", complete with hotel and even a post office.

The following selection is from Simpson's journal of 1842:

"Next day, being Monday the twenty-sixth, I started in the small steamer to visit some hot springs, which are used chiefly as baths, about twenty miles to the north [sic] of New Archangel, and arrived at my destination about three in the afternoon. The establishment in the neighborhood consisted of three snug cottages, being kept in order by an old fellow of a Russian and his daughter, both of whom, whether from choice or by way of example, took a plunge every day, for half an hour at a time. The damsel's rosy cheeks seemed to speak volumes in favor of the waters, though, perhaps, they were merely the result of being cooked every forenoon in a temperature of upwards of 130° of Fahrenheit. This establishment is employed as an hospital for invalids from Sitka, and is found to be efficacious in rheumatism, fevers, syphilis and cutaneous disorders, the process being to bathe in the first three of these four cases, and to drink in the last. The buildings are pleasantly situated on the sloping face of a bank at a distance of about a hundred yards from the sea. . . .

"In the neighborhood there are four distinct springs, all taking their rise from fissures in the granite rock, with which these islands abound. At the source the principal spring possesses a temperature of 54° of Reaumur, or 153°° of Fahrenheit, being hot enough, as we found by experiment, to cook an egg in eight minutes. . . .

"As this natural 'medicine' is held in high estimation by the surrounding tribes, the native bath enjoys no sinecure of it. When the country is sufficiently peaceable for moving about in safety, the savages think nothing of coming two hundred or three hundred miles to benefit by the healing waters, while they do their best to take their traveling expenses out of them by lying in soak for hours at a time with nothing but their heads visible, eating, drinking, and I may add, sleeping in the bath. I can certainly vouch to the extent of four hours for two women, one of them with an ulcer on her hip, and the other with an affection of the spine. When reduced to a state of lassitude by the parboiling, particularly after taking a meal in the caldron, the patients munch a little snow, when such a luxury is attainable, by way of dessert. . . .

"I remained only one night, having bathed twice. . . . I was glad to scramble out immediately as red as a lobster, or, to humor the locality in the way of a simile, as red as the rosy cheeks aforesaid of the lady of the ascendant. This tutulary nymph, by the by, led a recent visitor into a capital mistake. In her capacity of handmaid, she had frequent occasion to enter the room where he was sitting; and invariable, on coming into the presence, she bowed down to the ground at every step, crossing herself reverentially all the while. . . . In spite of my maturer years, the pretty maiden behaved towards me in the same extraordinary way; and what was far more mysterious, she still continued, when I slipped away to try her, to rehearse her obeisances before the empty chair. While I was speculating on her proceedings, I happened to cast my eye on the grim visage of an image, which occupied a niche in a dark corner of the room immediately behind the position which I had just left. The bubble was now burst; . . . [T]he naiad's conspicuous attentions had been directed over his head to a musty old saint."

REFERENCE

Simpson, Sir George: *An overland journey round the world during the years 1841 and 1842.* Two volumes (Philadelphia: Lea and Blanchard, 1847.) Vol. 2, p. 83.

From Out of the Past . . . Over Thirty Years Ago

Gloria K. Park, M.D.

Excerpts from a medical manual provided to village teachers in 1944. In other words, how to practice medicine in the pre-penicillin days. The manual was prepared by Dr. Fred M. Langsam of Bethel and used for several years until new guides were prepared by the Alaska Department of Health. For comparison on available drugs, take a look at the size of the current PDR, whose first edition was 1946.

MEDICAL MANUAL FOR TEACHERS

AUGUST 1944

Sore Throat — Always look at throat. White covering of tonsils extending onto soft palate and combined with difficult swallowing and foul breath — suspect diphtheria. Notify doctor.

Bronchitis — Cough with tickling or painful sensation in upper portion of chest. Wear warm jacket around chest (pneumonia jacket), stay indoors, take hot drinks. For productive cough (patient brings up much phlegm) give Elixir Terpin hydrate with Codeine — 1 to 2 teaspoons in half glass of water every 3 to 4 hours. For dry cough give Brown Mixture (mixture opium and Glycerrhiza) as above.

Grippe — Note: A cold can affect eyes, nose, throat, larynx, bronchial tubes alone or in any combination. Treat according to organ affected. If added to this there are general symptoms such as fever, loss of appetite, backache, suspect grippe. If many people suffer from grippe at the same time, suspect flu (influenza).

Pneumonia — Patient becomes ill suddenly, has cough with pain in side, may bring up rusty phlegm. Keep patient in bed in half sitting position to make breathing easier. Apply warm flannel jacket next to skin of chest (pneumonia jacket). Provide for easy bowel movements (soap suds enema best). Give sedative for sleeplessness — ° to 1 teaspoon of Elixir sodium bromide or 1 to 2 5-grain tablets of Potassium bromide (or any other 5-grain bromide tablet). Notify doctor. For treatment with Sulfa-drug, see paragraph at end of pamphlet.

Tuberculosis — Restrict activity. Provide for good nourishment. Cod liver oil. Isolation as far as possible. Do not make patient feel "unclean." Advise nearest physician or medical director.

Lung Hemorrhage — Calm patient. Few patients die of hemorrhage. Check individuals activity entirely during hemorrhage. Keep patient in bed in half-sitting position. Apply cold (ice bag, etc.) to chest on side from which bleeding comes. (Patient will know). Give sedatives — bromides. (See pneumonia.) Patient must not talk or whisper. Is not to eat solid food, but only cool nourishing liquids for 4 or 5 days. To stay in bed six weeks. Notify nearest physician.

Pulling teeth — Be sure to clasp tooth low down. Tips of forceps should squeeze in between tooth and gums.

Impetigo — Wash with soap and hot water, removing all scabs. Paint with mercurochrome, then apply ammoniated Mercury Ointment.

Note: Never use iodine together with Ammoniated Mercury. Repeat dressings daily. If scalp affected in children cut hair closely and then apply treatment as above.

Boils — Do not squeeze. Paint with disinfectant. Apply Ichthyol Ointment on dressing. Apply dry heat. When boil "points" (yellow spot appears on skin showing where pus is) dip pointed toothpick in Tincture of Iodine, then puncture yellow spot. After pus comes use hot wet Epsom Salt dressing (Tablespoonful to cup of hot water).

Earache — Put several drops warm glycerine or sweet oil in ear. Cotton plug, hot water bottle to ear, aspirin for pain.

Running ears — Wash with boric acid solution (1 teaspoon boric acid to 1 glass warm water). Use a rubber bulb syringe or enema set. Dry ear carefully. Drop 2 drops Mercurochrome, 3 drops glycerine, cotton plug. The plug must be changed frequently enough to prevent collection of pus in ear. Bad tonsils and adenoids frequently cause running ears. May result in deafness.

SULFA DRUGS

There are various sulfa drugs. One of the safest is sulfadiazine. However, there is still some danger connected with its use. Try to give it only under guidance of physician. In case of emergency you may use it in treatment of pneumonia and gonorrhea.

Never use Epsom Salt with sulfa drug. While patient is receiving sulfa drug he must drink much water. Signs that show the drug is poisoning — patient blood in urine, dizziness, nausea and vomiting. If you think that the drug may be hurting patient, discontinue immediately.

LISTS OF MEDICINES AND SUPPLIES AND SUGGESTED AMOUNT TO BE KEPT ON HAND

Brown mixture — 4 qts. 1 to 2 teaspoonsful in ° glass water 3 to 4 times a day for dry cough.

Elixir Terpine Hydrate with Codeine — 4 qts. 1 to 2 teaspoons in ° glass of water 3 or 4 times a day for productive cough and coughing of chronic tuberculous.

White Pine Sirup(sic) — 2 qts. - ° teaspoon diluted with a little water 2 or 3 times a day for cough in children.

Cod Liver Oil — 50 gallons - Infants 2 weeks old, 10 drops increase so that 1 teaspoon at 6 months and continue. Pre-school age, 2 teaspoons a day; school children, 1 tablespoon a day; adults suffering from tuberculosis, 2 tablespoons a day.

Blaud's Pills — 1,000 - 2 tablets 3 times a day after meals for anemia. Continue for 60 days. Stool may turn black. Guard against constipation.

Sodium Bicarbonate — 5 gr. tablets, 5,000, 2 to 4 tablets with water for sour stomach. Useful for indigestion during pregnancy.

Hinkle's Pills — 300 (same as Cascara Compound), 1 to 3 tablets at bedtime with much water against constipation.

Iodoform Powder — 4 1 oz. bottles - Dust on draining tuberculous sinuses. Also good for boils and foul smelling wounds.

Ammoniated Mercury — 10% - 5 pounds - Use against impetigo and infected superficial wounds.

Ichthyol — 10% - 2 pounds, use against boils.

Boric Acid — 10% - 5 pounds - Use against skin irritations, burns, etc.

Sulphur Ointment — 2 pounds - Use against scabies (itch).

Vaseline — 5 pounds - Use as lubricant and bland dressing.

Zinc Oxide — 2 pounds - Use against skin irritations and to protect skin surrounding sores.

Atropine Sulfate — 1% - 1 dozen tubes for eye conditions with squinting.

Sulfathiazole — 5% - 1 dozen tubes for eyes of newborn babies.

Yellow Oxide of Mercury — 1% - 2 dozen tubes for eye irritations. May be used together with Atropine Sulfate.

Boric Acid — 5% - 2 dozen tubes for chronic eye irritations.

Holocaine and Epinephrine — 1 dozen tubes - Use against snow-blindness.

Sulfadiazine — 7.7 gr. tablets - 1,000 - Use only as directed in this pamphlet.

Coryza tablets — 500 - For running nose and itching eyes in colds. One tablet every half hour until throat feels dry, then every 3 hours. May be used together with aspirin.

Methenamine (Same as Urotropine) — 200 tablets - 5 gr. Against bladder infections used under physician's direction.

Oil of Cloves — 1 oz. bottles - Use on cotton pledget in cavity of aching tooth.

Sodium Perborate — 1 pound - As mouthwash in mouth infections - 1 teaspoon in glass of water.

Chloramine Tablets — 1,000 - Use as gargle and mouthwash, 1 tablet in ° to 1 glass warm water.

Tr. of benzoin compound — For cracked lips and nipples.

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Quit Rates at One Year Follow-up of Alaska Native Medical Center Tobacco Cessation Program

Michelle R. Hensel[1]
Tim Cavanagh, M.D.[2]
Anne P. Lanier, M.D., M.P.H.[3]
Teri Gleason, R.N.[2]
Bill Bouwens, M.P.H.[2]
Harvey Tanttila[2]
Augusta Reimer, M.P.H.[2]
Roger L. Dinwiddie, Pharm.D.[2]
James C. Hayes[2]

ABSTRACT

The prevalence of tobacco use in the Alaska Native population is unusually high, as high as 50% in both adult men and women. In June of 1992, the Alaska Native Medical Center and the Alaska Area Native Health Service began a tobacco cessation program using behavioral modification classes and transdermal nicotine patches. Patients were subsequently followed at three month intervals for a year to assess smoking status. To date, 193 people have completed the program with at least three months having elapsed since completion of classes. The quit rates at three, six, nine, and twelve months were 31%, 30%, 24%, 21% respectively. The long-term quit rates for this tobacco cessation program are comparable to the rates of other studies which have included both behavioral modification and transdermal nicotine.

INTRODUCTION

Alaska ranks sixth highest in smoking prevalence among states in the U.S. (1). Within Alaska, the prevalence is higher among Alaska Natives (39-50%) than non-Natives (25%)(2), there being regional variation among the Native populations (3). High rates of cigarette use occur among Alaska Native children as well as adults (3). Some evidence indicates Alaska Natives begin smoking cigarettes at an earlier age than non-Natives (3). High rates of smokeless tobacco use are also found among Alaska Natives, especially children (4).

A high percentage of lung cancer cases are caused by cigarette smoking (5). Lung cancer is the most rapidly increasing cancer among Alaska Native people and other indigenous populations in the arctic (3). Incidence rates have increased several-fold during the past 30 years (3). Lung cancer mortality rates of Alaska Natives are higher than any other Native American group served by Indian Health Service (IHS) and exceed rates of the U.S. encompassing all races (6). Lung cancer mortality rates are the most rapidly increasing cancer mortality rates in the Alaska Native population. The rates are increasing more rapidly in women than in men (6). Reduction in cigarette smoking is the single most important way to reduce lung cancer and other tobacco-related diseases (5).

The term Alaska Native refers to Eskimos, Indians, and Aleuts whose ancestors originally inhabited the land now included in the state of Alaska. The IHS, tribal and Native regional health corporations provide health care to Alaska Natives. In June 1992, the Alaska Native Medical Center (ANMC) implemented a tobacco cessation program for Alaska Natives residing in the Anchorage area. The program combines behavioral modification classes as designed by either the American Cancer Society (ACS) or the American Lung Association (ALA) with the use of transdermal nicotine patches.

There are high relapse rates with any smoking cessation therapy. It has been shown that the transdermal nicotine patch significantly reduces tobacco withdrawal symptoms and improves smoking cessation rates (7-12). Use of the transdermal nicotine system has been shown to be more effective than behavioral therapy alone (9). Nevertheless, for optimal effectiveness the transdermal nicotine patch has been recommended for use in conjunction with a behavioral modification program (8,11). In a program combining the transdermal nicotine patch with behavioral therapy, the six month abstinence rate was 26% versUs 12% for behavioral therapy alone (7). The six month cessation rate using the nicotine patch without a behavioral component was 22% (13). Most studies provide data on quit rates for only a short interval after therapy. Long-term quit rates (>6 months), may be much lower than initial quit rates (13).

---

(1) University of Washington Medical School, Seattle, WA.
(2) Alaska Native Medical Center, Anchorage, AK.
(3) Alaska Area Native Health Service, Anchorage, AK.

Address reprints to: Anne Lanier, M.D., M.P.H., AANHS, 250 Gambell Street, Anchorage, AK 99501.

We report preliminary results for the ANMC tobacco cessation program; this program provides behavioral therapy and nicotine patches at no cost to participants.

METHODS

ANMC's program began in June, 1992. The availability of transdermal nicotine patches and the advertising promoting the product in 1992 prompted many patients to request prescriptions for patches. ANMC's pharmacy did not include nicotine patches. The Pharmacy and Therapeutics Committee and hospital administration were reluctant to make patches available without an existing behavioral cessation program. A program was initiated under the auspices of the Patient Education department at the ANMC. It was agreed that the program would be conducted for a designated period of time, data collected, and continuation of the program would be based on the results.

Patients were referred by ANMC providers to the Patient Education Department at the Medical Center. They were enrolled either in classes which consisted of four group counselling sessions over a two week period based on ACS's "Fresh Start" program or seven sessions over a six week period using ALA's "Freedom from Smoking" program. ACS classes were taught by the ANMC Tobacco Coordinator and ALA classes were conducted under contract at ANMC. At the beginning of the tobacco cessation classes, each patient filled out a questionnaire which included information concerning age, sex, smoking history, and ANMC employee status. Participants were given an appointment in the Family Medicine clinic which included a problem-specific history and physical examination, a determination of appropriate initial nicotine dose, and a prescription for the nicotine patches. Patches were given out after the second week of class (after class three for ALA and class four for ACS). Patients were classified as having completed the classes if they attended three of four ACS classes and at least four of seven ALA classes.

Patches were available in three initial dose strengths, to be tapered over time. They were given an initial prescription for four weeks of patches which could be renewed for up to fourteen weeks. A physician or pharmacist attended the class prior to initiation of transdermal nicotine patch use and provided pertinent drug information and instruction. The initial four week prescription was filled at that time; patients returned to the outpatient pharmacy for refills.

Prescriptions dispensed by the ANMC pharmacy are entered into a computerized database making it possible to quantitate the number of nicotine patches dispensed for each participant. In addition, at the time of follow-up, each participant was queried as to whether or not they had used patches and the quantity used. Those who participated in the classes were matched with those for whom patches were prescribed and the information on patch dosage and number of patches summarized.

All services and medications were provided at no charge to the patient. The program was offered only to adult IHS beneficiaries and/or ANMC employees. Patients were advised at enrollment that they would be contacted up to one year following the classes and were called by phone at three, six, nine, and twelve month follow-up intervals to assess smoking status. The timing of follow-up interviews was determined from the start date of behavior modification classes by the following means. Intervals were calculated from two weeks following the start date of ACS classes and four weeks following the start date of ALA classes which coincides with the end of the group counselling sessions. Since enrollment occurred continually from start of the program through May 1993, the maximum number of patients contributed to calculations of the three month quit rate. Successively fewer contributed to the six, nine and twelve month calculations. Some patients were unavailable at a specified follow-up interval. At the next interval, the interviewer queried about both current and recent past cigarette use to determine the status at each of the three month intervals. Factors associated with smoking cessation were analyzed at the six month interval using the chi square test and a statistical test for linear trend and proportions (14).

RESULTS

As of August 1993, 252 people had begun the classes and 193 people were 3 to 12 months post-completion of their classes. Therefore, 193 people were eligible for data collection at the three month follow-up interval, 111 people were eligible for the six month follow-up, 64 people were nine months post-completion, and 24 people were one year post-completion of the classes. Table 1 describes all 193 patients. Forty-six patients (24%) were ANMC employees. There were 2.4 times as many women as men. The majority of participants were 30-59 years of age, the 40-44 year group being the largest five-year category represented.

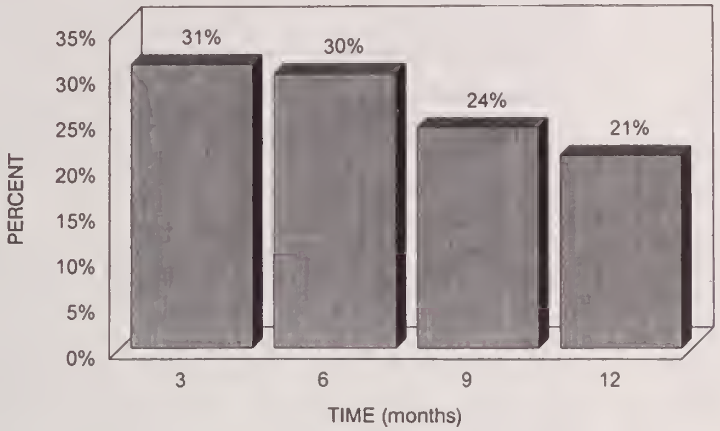

FIGURE 1. Tobacco quit rates for participants in behavioral modification/nicotine patch program at three, six, nine and twelve months after completion of the class.

Figure 1 shows quit rates at three, six, nine, and twelve months after class completion. At the three month follow-up date, 37 of 193 people could not be contacted. Of the 156 contacted, 49 (31%) stated they were no longer using cigarettes. At six months, 40 people were lost to follow-up. Of the 71 contacted, 21 (30%) reported abstinence from tobacco. At nine months, 9 people were lost to follow-up. Of the 55 contacted, 13 (24%) reported to have quit cigarettes at that time. Lastly, at one year, five out of 24 were lost to follow-up while four out of 19 (21%) reported cigarette abstinence.

Table 2 describes characteristics of the 71 patients contacted at six months after completion of the class. Data were analyzed for any significant differences between the quitters and the non- quitters with regard to smoking history, sex, age, and employee status, or packs smoked per day before classes began. Using a test for linear trend and proportions (14), we found no association between quit rates and the duration of smoking prior to beginning of class. However, we did find a higher cessation rate in the older age group (>45 years of age) which was not quite statistically significant (p=.055). The quit rate was 17%, 27%, and 42% for people ≤35, >35 and ≤45, and >45 years old, respectively. We did a subset analysis using the chi square test to see if this trend was related to the higher percentage of males in this older age category, who had higher overall cessation rates (p=.112). Our analysis showed this relationship was not significant.

Data on nicotine patch use were summarized to provide estimates of the average duration of patch use, dosage strength used and costs of the medications required for the program. Of the 193 participants who had completed classes and were at least 3 months post-completion, 180 had nicotine patch prescription data found in the computerized database. Therefore, 13 (7%) of the 193 participated in the tobacco cessation program without receiving a prescription for patches. An additional nine (5%) reported at follow-up that they had not used the patches they received from the pharmacy.

The 180 participants received a total of 545 boxes of nicotine patches; a box contains 14 patches or a two-week supply. Of those who used patches, 131 (73%) started on the 21 mg dose; forty-nine participants (27%) began at the 14 mg dose. Based on the prescription data, the minimum prescription was two weeks (1 box), while the longest was 14 weeks. However, patient interviews suggest that actual use may have been even less than prescribed (minimum one to two days, maximum 12 weeks).

TABLE 1.
Descriptive Characteristics of Participants who Completed the Tobacco Cessation Program

N=193

Non-Employee	Age	Female	Male	Total
	20-24	2	1	3
	25-29	6	5	11
	30-34	22	5	27
	35-39	17	9	26
	40-44	17	7	24
	45-49	12	7	19
	50-54	12	6	18
	55-59	8	5	13
	60-64	3	1	4
	65-69	1	0	1
	75-79	1	0	1
Subtotal		101	46	147
ANMC Employee				
	25-29	1	1	2
	30-34	4	1	5
	35-39	5	1	6
	40-44	6	3	9
	45-49	7	2	9
	50-54	6	2	8
	55-59	5	1	6
	60-64	1	0	1
Subtotal		35	11	46
Total		**136**	**57**	**193**

TABLE 2.

Descriptive Characteristics of Patients at Six Months Post-Completion of Classes

N=71

Patient status	Total	Quit	Non-Quit	Quit
Females	50	12	38	24%
Males	21	9	12	43%
ANMC Employee	30	9	21	30%
Non-Employee	41	12	29	29%
Packs per day <1	36	11	25	31%
Packs per day >1	35	10	25	29%
Years smoking cigarettes				
<15	11	2	9	18%
>15 <30	38	13	25	34%
>30	22	6	16	27%
Age (years)				
<35	23	4	19	17%
>35 <45	22	6	16	27%
>45	26	11	15	42%

DISCUSSION

The tobacco cessation program initiated at ANMC was evaluated after one year. The quit rates of 31%, 30%, 24%, and 21% at three, six, nine, and twelve months, respectively, are comparable to those of other studies. In studies using transdermal nicotine without concurrent behavioral therapy, Abelin et al, 100 subjects reported abstinence rates of 36%, 22%, 17%, and 18% at three, six, nine, and twelve months respectively (13). Tonnesen et al reported abstinence rates of 53%, 41%, 24%, and 17% at six, 12, 26, and 52 weeks respectively (8). There are a few studies done using concurrent behavioral therapy that report long-term quit rates. Daughton et al, in a study of 51 subjects, reported a six month cessation rate of 22% (13) while the Transdermal Nicotine Study Group reported a six month abstinence rate of 26% (7). Buchkremer et al, found quit rates of 36%, 34%, 28%, and 26% for patients evaluated at three, six, nine, and twelve months (15). Hurt et al, reported abstinence rates of 37%, 29%, 26%, and 26% for 18, 26, 40, and 56 weeks respectively (13).

There were a considerable number of people lost to follow-up in our program. We were unable to contact 19% of patients at three months, 36% at six months, 14% at nine months, and 27% at twelve months. The Alaska Native population is very mobile, moving between urban centers and home villages on a regular seasonal basis on account of jobs, family ties, commercial fishing and subsistence activities. Although smoking status was self-reported, the patients were found to be honest, open, and willing to discuss their experience. Because patients receive their health care almost exclusively at the ANMC, we will be able to document and verify smoking status at future clinic visits.

We did not find any significant differences between those who remained abstinent and those who resumed smoking with regard to age, sex, employee status, or smoking history. Other studies have shown conflicting results as to how the initial degree of nicotine dependence relates to the success of the transdermal nicotine patch in smoking cessation programs. Buchkremer et al, reported that light smokers had a higher tobacco cessation success rate than heavy smokers (9). Two different studies have shown that smoking history has no effect on patch treatment response (7,10).

Men had higher cessation rates than women in our study, however, this finding was not statistically significant. Another study had found that the nicotine patches are equally effective in men and women (13). In our review, patients in the older age group (>45) were found to have better cessation rates (p=.055). Many different reasons may explain this trend; perhaps people in this older age group have been advised to quit smoking for serious health conditions. Palmer et al found that quit rates for patients over 60 years of age were comparable to those for patients 60 years or less (13).

Data concerning nicotine patch use by participants in this tobacco cessation program indicate that all but a small percentage chose to use the patch. This was anticipated because it was clear to the program organizers that the demand for patches following company advertising of the patches prompted the initiation of the program. On the other hand, it is apparent from both computerized pharmacy data and patient interviews that some patients who filled prescriptions did not use a full course of treatment. The study design did not include questions which might shed light on the cause for this observation. Patches were not offered until the end of the third or fourth session; consequently some participants may have felt they could utilize newly acquired behavioral skills without the need for patches. Some participants did not require full strength patches, and many used the patches for a shorter duration than recommended by the manufacturer. These factors, along with a reduction in price of the medication after the beginning of the program, meant that the cost per patient enrolled in the program was actually less than originally anticipated.

After the first year of operation of the ANMC tobacco cessation program, preliminary results are very encouraging. Since the use of tobacco is so high in the

Alaska Native population, the fact that the cessation rates are comparable to those of other published studies supports the conclusion that the program has been very successful and should be continued. The Alaska Department of Public Health reports that the direct health care costs for treatment of smoking-related diseases in 1991 were \$941/year per current smoker $\geq$35 years of age (16). This figure does not include lost wages or health effects on children, spouse, and passive smoking. Our estimate for the first year of the program is approximately \$720 per quitter, and of course, much less per participant. Since ex-smokers often experience several unsuccessful attempts before successfully quitting, it is likely that all participants completing the program gained information and skills which may eventually assist them in permanent tobacco cessation. The cost of direct health care per smoker approximates the cost of the cessation program. The cost of this program per patient is less than the combination of direct and indirect health care dollars the state of Alaska spends per smoker. It is anticipated that program costs can be further reduced as the program gains support and popularity. Clearly, the cost of such a secondary prevention program is outweighed by both the anticipated decrease in medical costs related to long-term tobacco use and the more subjective benefit of improved health for Alaska Natives.

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Characteristics of Alaskan Family Physicians as Determinants of Practice Location

Jane MacGregor Potter, M.D.(1)

ABSTRACT

Characteristics of Alaskan family physicians were studied as determinants of practice location. A four-page questionnaire was mailed to 233 Alaskan family physicians. The background of the physicians and spouses had no significance in predicting future practice locations. Rural physicians were more likely than urban physicians to have had rural experience after entering medical school. Experience in a Third World country was not significant between rural and urban physicians. Significant factors for urban practice location were access to medical consultation, availability of CME, proximity of extended family/friends, employment/educational opportunities for spouse, cultural advantages, a salaried position, and children's educational opportunities. Factors significant for rural practice location were size of community, opportunity for subsistence hunting/fishing, and a feeling of being needed. Future practice location of Alaskan family physicians is not based solely on background prior to medical school, but on activities and attitudes shown predictive of practice location during medical training.

INTRODUCTION

The shortage of rural physicians is demonstrated in numerous studies. While data on Alaskan rural physicians supply is not well documented, data on the overall supply of Alaskan physicians is given in the volume *Health Care in Rural America* by Congressional Office of Technology Assessment (1). The ratio of non-federal Alaskan physicians in 1988 per 100,000 civilian residents was 145, with an 8.2% increase from 1980-1988. The only two states with lower ratios were Idaho and Mississippi, with 136 (17.2% increase) and 135 (20.5% increase) non-federal physicians per 100,000 residents respectively.

The Comprehensive Health Manpower Training Act of 1971(2) addressed the shortage of physicians in rural areas by authorizing grants to "establish and operate projects designed to identify and increase admissions to and enrollment in schools of medicine. . . of individuals whose background and interests make it reasonable to assume that they will engage in the practice of their profession in rural or other areas having a severe shortage of personnel."

Cullison et al. (3) concluded that a physician with a nonmetropolitan background (population less than 50,000) is two to three times as likely to practice in a nonmetropolitan area than physicians with an urban background. Cullison et al. also investigated family physicians, concluding that two-thirds of family physicians with rural backgrounds (population less than 50,000) enter nonmetropolitan practice, compared to one-fourth of family physicians with a metropolitan background (population greater than 50,000) who enter nonmetropolitan practice. Head and Harris (4) found that the size of the community of origin of the physician and spouse was important to practice site selection.

Numerous references cite medical education and residency location as major determinants in practice location (5-7); however, Alaska does not have a medical school or residency program except the state's connection with the WAMI (Washington, Alaska, Montana, Idaho) program at The University of Washington.

The effect of rural medical exposure was studied by Phillips and Swanson (8) who revealed that 64% of the University of Washington students who did a rural family medicine clerkship are either pursuing family practice or are in rural family practice.

Personal factors influencing practice location have been studied and found to be statistically significant between urban and rural populations. Gessert et al. (5) found the following factors to be significant to urban physicians: access to medical consultation, opportunity to practice in a salaried position, and proximity of extended family and friends. Significant factors to rural physicians were size of community and amount of crime. Leonardson et al. (9) found the number of contacts with other physicians, cultural opportunities, and CME opportunities significant to urban physicians.

Rural background is used by medical schools to predict future practice location of applicants. Michigan State University (10) and Jefferson Medical College (11) have established programs to increase the number of rural physicians in their states based on preferential admissions of applicants with rural backgrounds. Without further research, the preferential admission of applicants based on rural background should be questioned. The hypothesis is that, 1) experience in rural medicine during training is more significant in predicting practice location than rural background, 2) expereince in Third World countries will positively impact future rural practice, and 3) there are significant rural and urban differences in personal factors that influence physician practice location, as described in earlier studies.

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(1) University of Washington School of Medicine, Seattle, WA 98195.

METHODS

A questionnaire was designed to examine the characteristics of Alaskan family physicians as determinants of practice location from the literature and discussions with Alaskan physicians. The four-page questionnaire was pretested by twelve Seattle family physicians and seven rural family physicians from Omak, Washington. The revised questionnaire was sent to all 233 family and general practice, federal and non-federal, MD's and DO's in the 1992 Alaska State Medical Association Medical Directory. A first mailing response rate of 117/233 (53.9%) was obtained, with 156/233 (71.9%) questionnaires returned after a second mailing.

Based on the volume of data generated by the questionnaire, only selected aspects will be included in this paper. Demographics, rural exposure, and factors influencing practice location of family practice physicians are discussed in this paper.

The demographics section serves to describe the physician population surveyed by gender, age, race, marital status, number and age of children, and perceived educational opportunities of children in a rural community compared to an urban community.

Rural exposure is defined in the questionnaire as an individual or spouse having lived in a rural community, rural medical exposure during training, or Third World country experience. The rural medical exposure data includes time working in medical school, residency, as a physician, and other activities during medical training in communities of 20,000 people or less. The community size question asks the physician to code the size of community they were living in during four time periods of their life prior to medical school.

The section practice location examines factors that influenced a physician's current practice location. The factors are rated on a scale of one to five, with one being most important to the physician.

Communities were grouped into three community sizes, with Anchorage considered urban (53 physicians), Fairbanks and Eagle River between urban and rural (19 physicians), and all other communities rural (84 physicians). The data was analyzed using Chi-square and T-tests.

RESULTS

Demographics: Two-thirds (100/150) of the total physicians were male, with one-half (53/100) of those in rural communities. The remaining one-third (50/150) physicians were female, with two-thirds (30/50) of those in rural communities. The majority of physicians were under the age 50: 67.9% (36/53) of Anchorage physicians and 85.6% (71/83) of rural physicians. Caucasians represent 89.6% of Alaskan physicians. Rural communities had the highest percentage of minority physicians (Asian, Black, Hispanic, Native American, and other) with 13.2%, compared to a 7.5% minority rate in the urban city of Anchorage. Most physicians were married (69.5%), with rural communities having the highest rate of single physicians, 19.3%, compared to 13.2% in Anchorage.

Rural physicians had more children under college age, with 1.2 children per physician (98/83) compared to urban physicians with 0.94 children per physician (50/53). Thirty-seven percent (30/81) of rural physicians felt that their children would have a better education in an urban environment compared to 45.7% (37/81) who disagreed and felt that their children are getting just as good an education in a rural environment. The 14

Table 1.

Physician Background vs Practice Location Size

Age Group		Anchorage	Fairbanks	Rural	Totals
Birth-7 yrs:	rural*	16 (32.0%)	6 (12.9%)	28 (56.0%)	50 (32.7%)
	urban	37 (35.9%)	11 (10.7%)	55 (53.4%)	103 (67.3%)
Totals		53	17	83	153
8-13 yrs:	rural	17 (34.0%)	7 (14.0%)	26 (52.0%)	50 (32.7%)
	urban	36 (35.0%)	10 (9.7%)	57 (55.3%)	103 (67.3%)
Totals		53	17	83	153
14-18 yrs:	rural	16 (35.6%)	6 (13.3%)	23 (51.1%)	45 (29.2%)
	urban	37 (33.9%)	12 (11.0%)	60 (55.1%)	109 (70.8%)
Totals		53	18	83	154
> 18 yrs:	rural	6 (17.7%)	2 (5.9%)	16 (47.1%)	24 (15.7%)
	urban	47 (36.4%)	15 (11.6%)	67 (51.9%)	129 (84.3%)
Totals		53	17	83	153

*rural = <20,000 people not adjacent to metropolitan area (>100,000 people)

Table 2.

Spouse Background vs Current Community Size

Age Group		Anchorage	Fairbanks	Rural	Totals
Birth-7 yrs:	rural*	18 (43.9%)	2 (4.9%)	21 (51.2%)	41 (32.5%)
	urban	27 (31.8%)	14 (16.5%)	44 (51.8%)	85 (67.5%)
Totals		45	16	65	126
8-13 yrs:	rural	15 (42.9%)	3 (8.6%)	17 (48.6%)	35 (28.0%)
	urban	29 (32.2%)	13 (14.4%)	48 (53.3%)	90 (72.0%)
Totals		44	16	65	125
14-18 yrs:	rural	14 (46.7%)	4 (13.3%)	12 (40.0%)	30 (23.8%)
	urban	30 (31.3%)	13 (13.5%)	53 (55.2%)	96 (76.2%)
Totals		44	17	65	126
> 18 yrs:	rural	6 (37.5%)	2 (12.5%)	8 (50.0%)	16 (12.7%)
	urban	38 (35.9%)	14 (13.2%)	58 (54.7%)	110 (87.3%)
Totals		44	16	66	126

*rural = <20,000 people not adjacent to metropolitan area (>100,000 people)

remaining physicians felt that the question was non-applicable and probably do not have school-aged children.

Rural Exposure: Background had no predictive value for future rural living, as 50% of physicians and spouses living in rural communities had urban backgrounds and 50% had rural backgrounds. (Tables 1 and 2).

Physicians living in rural communities had 100.9 months of rural exposure per person compared to urban physicians with 72.5 months rural exposure per person. (P<.05) Rural exposure looks at experience obtained by a physician in a community of less than 20,000 people during medical school, residency, as a physician, and other activities during medical training. Rural physicians had 22.1 months of Third World country experience per person compared to urban physicians with 26.3 months per person. That was not statistically significant.

Practice Location: Factors significant for rural practice location were the size of community, subsistence hunting/fishing (both above P<.01), cultural advantages, a salaried position, and children educational opportunities (all above P<.05). The level of debt from student loans was not statistically significant. (Table 3)

DISCUSSION

Alaska has a shortage of physicians relative to other states(1) and is at a disadvantage for physician recruitment without a medical school or residency program within the state (5-7). In an attempt to increase the number of rural physicians in Alaska, an applicants' background may be examined to predict whether that individual will eventually practice in a rural community. The questionnaire was developed to describe Alaskan physician demographics, examine the impact of rural background on practice location, examine the impact of rural medical exposure during training on practice location, and to indicate personal factors that influenced physician practice location.

Demographics: Rosenblatt et al. (12) examined demographics of medical students who entered rural practice between 1976 and 1985 and found that 23% of medical students were female, with over one and a half times more males entering rural practice. I found that one-third of Alaskan physicians are female, two-thirds of them in rural practice, compared to one-half of the males in rural practice. Rural Alaskan physicians are more likely than urban physicians to be under the age of 50, a minority, single, and have more children. Most rural physicians felt that their children were getting just as good an education in a rural community than an urban community.

Rural Exposure: Comparison between physician or spouse background and practice location detected no difference. Approximately one-half of the physicians and spouses with either a rural or urban background were found to enter rural practice regardless of background. My findings differ from those of Cullison et al. (3) and Head and Harris (4) and may reflect factors unique to Alaskan physicians, such as the lure of experiencing Alaskan wilderness regardless of physician background.

Experience throughout medical training plays a larger role in a physician choosing rural practice than physician background. My data concurs with Phillips and Swanson (8), as Alaskan rural physicians had more rural experience during medical training per physician, 100.9 months, compared to urban physicians with 72.5 months. (P<.05) There was no difference in Third World country experience of rural and urban physicians, which seems unusual as past interest in cross-cultural medicine would intuitively indicate future interest in cross-cultural medicine working with Alaskan Natives.

Practice Location: Factors influencing the practice location of Alaskan physicians are similar to those found by Gessert et al. (5) and Leonardson et al. (9). The following practice location variables are significantly important to urban Alaskan physicians: access to medi-

Table 3.

Factors Influencing Practice Location Selection

The numbers represent the mean on a scale of 1 to 5 with 1 being most important and 5 being least important.

	Urban Docs	Rural Docs
Factors Significant to Urban Physicians:		
Access to medical consultation	2.39 (n=51)	3:00 (n=95)**
Availability of CME	3.18 (n=51)	3.61 (n=94)**
Proximity of extended family/friends	3.22 (n=50)	4.00 (n=92)**
Spouse job/educational opportunities	2.86 (n=49)	3.45 (n=91)**
Cultural advantages	2.65 (n=49)	3.00 (n=93)*
A salaried position	3.19 (n=48)	3.66 (n=91)*
Children educational opportunities	2.53 (n=47)	3.01 (n=89)*
Factors Significant to Rural Physicians:		
Size of community	2.65 (n=51)	2.04 (n=97)**
Subsistence hunting/fishing	3.88 (n=48)	3.27 (n=93)**
A feeling of being needed	2.28 (n=50)	2.00 (n=97)*
Factor Significant to Neither Urban or Rural Physicians:		
Level of debt from student loans	4.37 (n=49)	4.42 (n=92)

**Significant at P< .01
* Significant at P< .05

cal consultation, availability of CME, proximity of extended family/friends, employment/educational opportunities for spouse, cultural advantages, a salaried position, and children's educational opportunities. Practice location factors significantly important to rural Alaskan physicians are size of community, opportunity for subsistence hunting/fishing, and a feeling of being needed. The level of debt from student loans was not a significant factor in choosing practice location.

Future practice location of Alaskan family physicians is not based on background prior to medical school. My hypothesis was supported by this study. Activities and attitudes during medical training are more predictive of practice location. The activities were rural exposure during medical school, residency, working as a physician, and other activities during medical training shown significantly greater in rural physicians. The attitudes that influenced rural Alaskan physicians in practice location were the size of the community, opportunity for subsistence hunting/fishing, and a feeling of being needed.

None of the cross-sectional studies on physician background and future practice location can determine the association between physician background and long-term rural retention that cohort studies could prove. Rural retention is the key to increasing rural physicians and requires further study.

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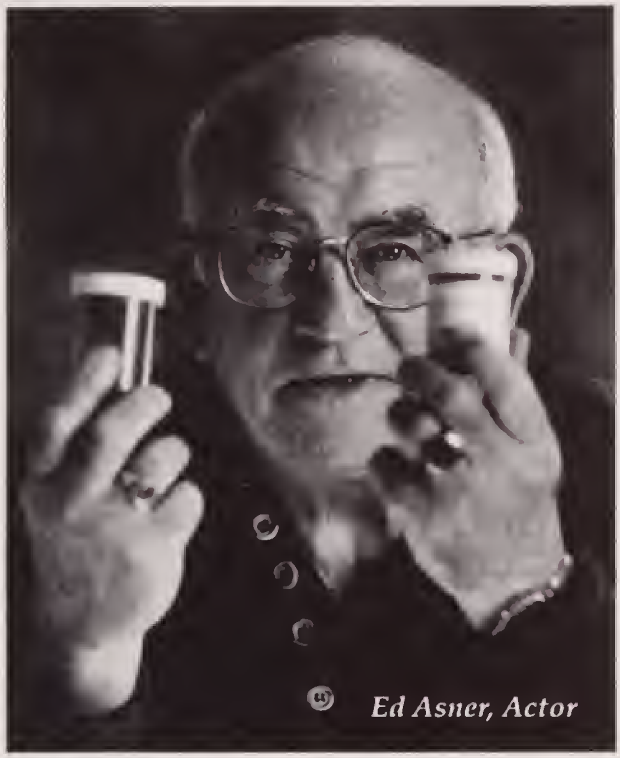
(continued on page 79)

Attention: Physicians



Ed Asner, Actor

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A public service message from the National Council on Patient Information and Education (NCPIE) and the U.S. Administration on Aging

Genetics of Breast Cancer - An Update

Anne P. Lanier, M.D., M.P.H.(1)
Stephanie D. Kaplan, BS(1)

The much anticipated breast cancer gene, BRCA1, was sequenced in October of 1994 (1). This discovery has endless implications for the fields of genetics and medicine and for individuals in hereditary breast cancer families. Breast cancer is the leading cancer in women; women in the United States have a one in eight chance of developing cancer if they live a full life span. Although rare in men, breast cancer is the second most frequently diagnosed cancer in both sexes combined. There are a total of approximately 575,000 cancers in women in the United States each year of which 182,000 are breast and 26,000 are ovarian (2).

The American Cancer Society provides yearly estimates, by state, on newly diagnosed cancers. In Alaska, it is estimated that 600 women will develop cancer each year, 130 of these will be breast cancer. Data from the Alaska Native Tumor Registry for the most recent four years (1989-1992), found that there were nearly 400 newly diagnosed invasive cancers in Alaska Native women, approximately 90 of these were breast cancer, 20 were ovarian cancer.

Cancer cases are generally placed in one of three categories: sporadic, familial or hereditary. Sporadic breast cancer is that which occurs randomly in the population. Familial cancer describes families with one or more relatives with the same cancer. Although familial clustering could be due to: chance, common exposure to an environmental cause, or genetics, first-degree relatives in these families are at two- to three-fold increased risk of developing the same cancer.

Hereditary breast cancer (HBC) and hereditary breast/ovarian cancer (HBOC) are terms used for multi-case families in which the family pedigree demonstrates genetic predisposition to the disease, particularly autosomal dominant inheritance. The hereditary forms of breast cancer account for about 5% of all breast cancer cases (1).

Extensive epidemiologic studies have identified risk factors for cancer of the breast. As for all cancers, age is a strong risk factor. Family history is the strongest of all other known risk factors (3). The magnitude of the risk depends on the degree (closeness) of the relationship, the number of relatives with a history of breast cancer and the age of diagnosis of the cancer. Most risk factors for ovarian cancer are similar to those of breast cancer (4), including age at menarche, age at menopause and nulliparity. Having cancer of the breast or ovary also increases the risk for developing the other cancer, ovarian or breast, respectively(5).

Table 1.

Cumulative Risk of Second Breast Cancer

Age Group (years)	Cumulative Risk
30-39	0.33
40-49	0.50
50-59	0.60
60-69	0.65

As stated above, epidemiologic studies were able to quantify risks for cancer in family members of hereditary breast cancer patients before successful mapping and sequencing of the gene (6,7). The probability an individual will develop the disease during a specified period of time, for example in a lifetime, is the cumulative risk. The risk of a family member getting breast cancer is dependent on the number of relatives with the disease and the age at which those relatives were diagnosed (8,9). A woman with three first degree relatives with the disease has a 20% risk of developing breast

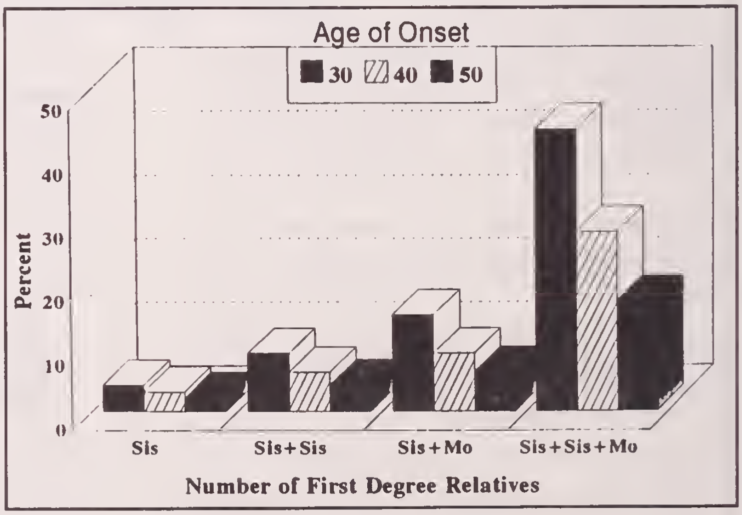

Figure 1. Risk of breast cancer with a family history of breast cancer in first degree relatives.

---

(1) Alaska Area Native Health Service, 250 Gambell Street, Anchorage, AK 99501.

Table 2.
Cumulative Risk of Breast and Ovarian Cancer in BRCA1-Gene Carriers

		Cumulative Risk of	
Age (years)	Breast Cancer	Ovarian Cancer	Either Cancer
30-39	0.032	0.0017	0.034
40-49	0.191	0.0061	0.195
50-59	0.508	0.227	0.619
60-69	0.542	0.298	0.678
70-79	0.850	0.633	0.945

cancer in her life if the relatives were 50 or older at the time of onset. Lifetime risk increases to 40% if any of the affected relatives were diagnosed in their 30s (8) (Figure I). Risks also increase if one has second degree relatives with the disease, but are not as high as the risks with affected first degree relatives. A person who has already had breast cancer is also at increased risk of cancer in the second breast (5) (Table 1). Individuals with a personal or family history of ovarian cancer are also at an increased risk for breast cancer(10). Members of HBOC families have a greatly increased risk of breast or ovarian cancer, or both. The lifetime risk for developing at least one of these cancers in BRCA1 gene carriers in families with evidence of linkage approaches 95%(5). Specifically, by age seventy the risk of breast cancer is 85% and the risk of ovarian cancer 63% in these gene carriers (5) (Table 2).

It is possible to estimate individual risks for concerned patients with family history of breast cancer. Two models have been published. One model bases calculations on the number and relationship of relatives with the cancer and the age at onset (8,9). This model does not include risk factors other than family history.

Another model estimates risk for patients with or without a family history of cancer (11). This model uses the following risk factors: age at menarche, age at first birth, nulliparity, age at menopause, as well as family history (number of first degree relatives). These estimates are the most specific individualized risk assessments available.

There are other genetic conditions, fortunately rare, in which there is an increased risk of breast cancer as well as other cancers and conditions. These syndromes include: Li-Fraumeni, Cowden's, Muir-Torre, Peutz-Jeghers and ataxia-telangiectasia (12,13). Table III summarizes clinical manifestations, genetic mutation and mode of inheritance for these syndromes (12) (Table 3).

Modern genetics and molecular biology have now confirmed the evidence for hereditary breast cancer (HBC) and hereditary breast and ovarian cancer (HBOC). As stated above, the majority of breast cancer is sporadic, however, HBC/HBOC may account for at least 9,000 cases of breast cancer a year. The following is a review of the recent genetic findings with regard to these hereditary cancers.

BRCA1 was isolated in October of 1994, after having originally been localized to chromosome 17q-12 in 1990 (14). Various mutations of this gene have been found through genetic analysis of families with extensive histories of breast and ovarian cancer. To date, thirty eight distinct mutations have been reported (15). The sequenced mutations include base pair insertions, base pair deletions, stop codons and missense substitutions (1). As we know, a single base pair change, insertion or deletion can produce a defective protein product. The BRCA1 gene's normal function is that of a tumor suppressor (16), hence any alteration in the gene, leading to a change in the protein product, may retard or eliminate its suppressor function. This causes uncontrolled cell proliferation leading to cancer.

A second breast cancer gene has also been localized, aptly named BRCA2 (17). It has been localized to chromosome 13, but has yet to be isolated. BRCA1 appears to account for less than half of the hereditary breast cancer, but for up to 80% of hereditary breast/ovarian cancer (HBOC). BRCA2 accounts for another 45% of hereditary breast cancer, but no hereditary breast/ovarian cancers. There is conflicting evidence stating what role (if any) the BRCA1 gene plays in sporadic cancer (16,18). Since BRCA1 and BRCA2 do not explain all hereditary breast and breast/ovarian cancer, other genes are proposed to exist.

The consequences of having sequenced a breast cancer gene are multi-fold and many questions have yet to be addressed. It eventually will be possible to test for the gene(s) in multicase families to determine if an individual is actually at an increased risk or if she does not carry the gene and is, therefore, at the same risk as the rest of the population. Men in the family may also want to know if they are carriers because of the importance of this to their daughters. When testing becomes available for presymptomatic individuals it is recommended that DNA testing be done only in a controlled setting with coordinated research and counseling (19). Identifying carriers of the genes could spare some patients unnecessary prophylactic surgeries, mastectomy and oophorectomy, and intensive early and aggressive screening. At the same time it could identify those who would be in need of enhanced screening. Although the uses for DNA testing are under intense discussion, the tests are not yet commercially available. A test for BRCA1 may be available in 1996. Development of a test for the BRCA2 is contingent on first isolating the gene.

Table 3.

Genetic Conditions Associated with Breast Cancer

Condition	Clinical	Mutation	Inheritance
Li-Fraumeni	Br,S,Bra,L,Adr	p53, 17p	Autosomal Dominant
Cowden's	Mucocutaneous lesions Benign Prolif disease Br,Thyr,Col		Autosomal Dominant Unknown
Muir-Torre	Benign Breast GI,GU,Skin,Br	MSH2, 2p MLH1, 3p	Autosomal Dominant
Peutz-Jeghers	Melanin GI polyps GI,Br,U,O,Tes	Unknown	Autosomal Dominant
AT	Ataxia telangiectasia L,Ly,Br,other	Unknown, 11q21	Autosomal Recessive

Legend: Br = breast, S = sarcoma, Bra = brain tumors, L = leukemia, Adr = adrenocortical carcinoma, Thyr = thyroid, Col = colonic neoplasms, GI = gastrointestinal, GU = genitourinary, U = uterua, O = ovary, Tes = testis, Ly = lymphoma.

In the mean time, it will be crucial to determine accurate family histories to identify those who will eventually benefit from DNA testing. Family histories should include at least three generations, with equal emphasis on paternal and maternal sides. Older second degree relatives may give more information than young first degree relatives because they have gone through the cancer age. Histories should include current age for living relatives, and age and accurate cause of death for those deceased (13). Identification of all family members who had cancer is especially critical. Information about these relatives should include cancer site, age at diagnosis and confirmation of the cancer diagnosis through medical records. Death certificates are less reliable, but are a source of information. Of course, permission must be obtained to contact relatives and each family member will have to sign for release of his or her medical records to obtain confirmation of the cancer diagnosis. Lastly, the interviewer should query for any children who may have had cancer as a clue to the rarer cancer family syndromes. This information will be critical in providing the data necessary to answer questions about appropriate surveillance and intervention measures.

DNA banks are being established for storage of biological samples of family members to assure samples will be available when testing becomes an option. Ideally samples from all affected family members would be available for testing to determine carriers and risks in unaffected relatives. Once hereditary cancer has been determined, a family should be entered into registries at major centers specializing in studies of high risk families. Registration would be advantageous to families in terms of comprehensive counseling and case management by a team including surgeons, oncologists, nurses, geneticists, psychologists, etc. It is also possible that testing may first be made available to these registered families on a research basis, although at this time registrants can not be guaranteed testing. It is anticipated that all comprehensive cancer centers in the near future will be prepared to provide cancer genetic counseling.

An important issue with respect to DNA testing is the need for, and availability of, counseling (12,20-24). Explaining the implications and results of complex genetic testing is difficult, but necessary. In order to assist with the interpretation of results, the provider should understand the patient's knowledge, understanding, experience and beliefs surrounding cancer in general, and breast cancer in particular. Patients must give informed consent for testing and must realize the influence the results could have on insurability. They must also be aware that absolute confidentiality of records can not be guaranteed. Each individual must make his or her own decision about testing, but in the case of hereditary diseases, results can have important ramifications for the whole family. Counseling must include not just the patient but the entire family. Counseling will be necessary in the following situations: choosing whether or not to be tested; understanding the magnitude of the risk; managing fears and anxiety in those who are at increased risk; and making decisions regarding prophylactic surgeries and other prevention options..

Counseling and management of these patients and families will be a long term process as the ages and risks of the patients increase and change. New tests, knowledge, risk estimates and options will also become available with time. Persons involved in counseling cancer families should have knowledge and experience in cancer genetics. There is only one certified genetics counselor in Alaska. She and others at the Alaska Genetics Clinic, Anchorage, Alaska have had some requests and experience in counseling cancer families. Although the Alaska Genetics Clinic deals primarily with perinatal counseling, they are willing to accept referrals for genetic counseling for cancer families as time allows.

Once those at increased risk in HBC and HBOC families are identified, more frequent screening should be implemented. Screening should include: breast self exams, clinical breast exams, mammography and transvaginal ultrasound. It is important to emphasize that although screening for breast cancer with breast examination and mammography has been proven efficacious, the efficacy of screening for ovarian cancer is not known. Some recommend screening high risk family members at six month intervals as early as age 25. Others recommend beginning cancer education and screening 5 to 10 years prior to the youngest age of diagnosis of breast or ovarian cancer in family members. This could mean teaching self breast examination to teenagers. Although prophylactic surgery is a consideration, many such decisions are being delayed until DNA testing for the BRCA genes is available.

These interventions (earlier screening, lifestyle modification and prophylactic surgery) are the only available options for high risk relatives. Not only are interventions limited, but they have yet to be proven effective in high risk families.

An option for these family members is involvement in the Breast Cancer Prevention Trial, the tamoxifen chemoprevention trial for breast cancer. It is still possible to enroll in this trial if the patient meets the selection criteria (25). Information on this trial, future prevention trials, the BRCA genes, and cancer in general can be obtained from the National Cancer Institute's Cancer Information Service. This and other sources of information are listed in the Appendix.

In conclusion, it is important to identify members of high risk families. This is crucial now as well as in the future when gene testing becomes available for breast cancer. High risk relatives should be identified in order to counsel patients about their risks, advise them about early detection, and keep them informed about new findings and intervention options in the field of genetics and breast cancer.

APPENDIX

National Cancer Institute
Cancer Information Services
CancerNet
1-800-4CANCER
CancerFax: 301-402-5877

Alaska Health Sciences Library
907-786-1870

Genetics Northwest (newsletter)
Pacific Northwest Regional Genetics Group
CDRC-Clinical Services Building
901 East 18th Avenue
Eugene, Oregon 97403-5254
Tel: 503-346-2610

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A Bibliometric Analysis of Cancer Among American Indians & Alaska Natives, 1966-1993

Martin C. Mahoney, M.D., Ph.D.(1)
Arthur M. Michalek, Ph.D.(1)

ABSTRACT

A bibliometric analysis was employed to objectively assess scientific studies published between 1966 and 1993 which have described cancer among American Indians and Alaska Natives. Searches of the MEDLINE (1966-1993) and CANCERLIT data bases (1983-1994) were used to identify relevant publications. In addition to examining publication sources and quantitative temporal trends, further bibliometric analyses were completed by considering a subset of papers published between 1982 and 1992. A total of 128 studies of cancer among American Indians and Alaska Natives were published between 1966 and 1993; 62 of these articles (48%) appeared between 1988 and 1993. Nine journals accounted for 53% of the total publications. The subset of 68 papers published between 1982 and 1992 were cited a total of 388 times in 136 different journals; the median number of citations was 2. Results demonstrate a limited number of published papers on cancer among American Indians and Alaska Natives. It is hoped that this paper will increase the awareness of cancer as an important health problem among American Indian and Alaska Natives and thereby serve to stimulate additional cancer-related research activities involving these groups.

Bibliometric analysis is emerging as an objective means to evaluate published research and to assess productivity in specific research areas.(1) The basis of bibliometrics is a quantitative assessment, that is the number of publications and an analysis of citation frequency and source. While the peer review process solicits input from a limited number of persons, an examination of citation frequency can be considered to represent the opinions of the larger body of peers working within the same general research area.(1) Although not considered to represent an absolute measure of scientific importance, citation frequency provides a parameter to assess the quality and impact of a research paper. Negative citations (e.g. criticizing or points out errors) and self-citations are thought not to distort citation analysis to a meaningful degree.(1)

Cancer represents an increasingly important health problem among American Indian and Alaska Native populations.(2-4) Individual research studies of cancer among American Indian & Alaska Native populations have appeared in the scientific literature. Justice (1989) (5) prepared a bibliography of cancer in American Indians and Alaska Natives during the period 1800-1989, however the data sources for this work were not described. Recently Owen et. al. (6) published a extensive bibliography of North American Indians and the health-related scientific literature covering the years 1950-1988. A meta-analytic approach has also been utilized in an attempt to summarize several studies of cancer incidence among Native populations.(7) To date, an objective analysis of the scientific literature on cancer among American Indians and Alaska Natives has not previously been attempted.

This manuscript presents a bibliometric analysis of studies of cancer morbidity and mortality among American Indians and Alaska Natives published between 1966 and 1993. This analysis considers the extent to which this research area has been featured in the scientific literature, sources of information on this topic and the frequency of citations as objective parameters of the breadth and importance of this research area.

METHODS

A MEDLINE (8) search was completed for relevant articles published between 1966 (initial year available on CD-ROM) and 1993. Search terms included: "Indi-

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(1) Departments of Educational Affairs & Natural Sciences, Roswell Park Cancer Institute, Buffalo, New York.

Address for correspondence:
420 Research Studies Center
Roswell Park Cancer Institute
Elm & Carlton Streets
Buffalo, NY 14263
phone: 716-845-2339 FAX: 716-845-8178

ans, North American," "Alaska," "Eskimos" including Inuit, Inupiat, Yupik and Aleut, "neoplasms," "cancer" and "cancer control and prevention." CANCERLIT included studies published between 1983 (initial year) and May 1994. Results from the MEDLINE search were supplemented by citations appearing in the CANCERLIT (9) data base using the same search strategy. While MEDLINE and CANCERLIT may not include all papers on this topic, these two sources include citations from an extensive number of high quality, peer-reviewed journals which have each satisfied specific criteria for inclusion in these data bases. Non-English citations as well as articles on South American Indians and Greenland Eskimos (Inuit) were excluded. Moreover, articles which appeared to be extraneous and/or to have included American Indians and Alaska Natives with other general population groups were also excluded.

In addition to examining publication sources and temporal trends in quantity, further bibliometric analyses of these cancer-related publications was completed by considering a subset of papers published between 1982 and 1992 (n=68); meeting abstracts and papers by anonymous authors were excluded fromthis sub-analysis. The choice of 1992 as the most recent year for this sub-analysis was made to allow for a time lag between publication of an article and its circulation, recognition and eventual incorporation into subsequent research. To assess citation frequency in this subset of papers, the Science Citation Index (SCI) was reviewed between 1985 and 1992, years for which this database was available on CD-ROM.(10) Each year the SCI includes more than 600,000 article citations appearing in more than 5,000 scientific journals.

RESULTS

Computerized searches of the MEDLINE & CANCERLIT data bases identified a total of 128 studies of cancer among American Indians and Alaska Natives, published between 1966 and 1993 (see Figure 1); 62 of these articles (48%) appeared between 1988 and 1993. The 17 articles published during 1993 were largely accounted for by proceedings from the national conference on Cancer in Indian Country held in 1992. In addition, these 128 articles included four abstracts published in conference proceedings, two book chapters, two letters, two editorials and a review article; thus, yielding a total of 117 full length manuscripts.

These 128 articles appeared in 55 unique publications. As presented in table 1, nine journals accounted for 53% of the total publications with the largest proportions of papers published in *Alaska Medicine* (11.7%) and *Journal of the National Cancer Institute* (10.2%).

Table 1.
Scientific journals publishing articles on cancer among American Indians & Alaska Natives, 1966-1993 (n=128[1]).

journal	number	percent
Alaska Medicine	15	11.7%
Journal of the National Cancer Institute	13	10.2%
Cancer	9	7.0%
International Journal of Epidemiology	7	5.5%
American Journal of Public Health	6	4.7%
Canadian Medical Association Journal	6	4.7%
Arctic Medical Research Journal	4	3.1%
National Cancer Institute Monographs	4	3.1%
Public Health Reports	4	3.1%
other	60	46.9%

Results of the bibliometric analyses are summarized in tables 2 and 3. The subset of 68 papers published between 1982 and 1992 were cited a total of 388 times in 136 different journals; the median number of citations was 2. Twenty papers (29%) were not subsequently cited, 23 papers (34%) yielded between 1 and 5 citations and 11 papers generated between 6 and 10 citations. The top five citing

Table 2.
Bibliometric analysis of published articles on cancer among American Indians & Alaska Natives, 1982-1992 (n=68[1]).

publication year	# published papers[1]	# of citations[2]
1982	4	
1983	5	
1984	5	
1985	7	12
1986	4	14
1987	4	18
1988	6	29
1989	11	52
1990	6	49
1991	6	59
1992	10	69
1993		86
total	68	388

[1] articles published 1982-1992, identified by MEDLINE & CANCERLIT search; excluding meeting abstracts and papers by anonymous authors.
[2] based on Science Citation Index on CD-ROM, 1985-1993.

journals accounted for 34.5% of the total citations: *Journal of the National Cancer Institute* - 38 citations (9.8%), *American Journal of Public Health* - 31 citations (8.0%), *International Journal of Epidemiology* - 30 citations (7.7%), *Cancer* - 24 citations (6.2%) and *Public Health Reports* - 11 citations (2.8%).

Table 3
Number of citations for published articles on cancer among American Indians & Alaska Natives, 1982-1992 (n=68[1]).

total citations[2]	# published papers	percent
none	20	29.4%
1-5	23	33.8%
6-10	11	16.2%
11-15	8	11.8%
16+	6	8.8%

mean: 5.7 citations
median: 2 citations
range: 0-37 citations

[1] articles published 1982-1992, identified by MEDLINE & CANCERLIT search; excluding meeting abstracts and papers by anonymous authors.
[2] based on Science Citation Index on CD-ROM, 1985-1993.

DISCUSSION

Since the present paper considered a specific area of scientific research rather than publications from a single journal, standard bibliometric parameters which are typically used in that context (e.g. the impact factor, the cited and citing half-lives and the immediacy factor) could not be utilized.(11) Instead, the overall or actual impact of a paper was determined based on the number of times the manuscript was subsequently cited. Citation frequency is a straightforward and readily interpretable method by which to assess research productivity and contributions.(1)

Citation frequency can be skewed by authors' self-citation, journal circulation size, time period since publication and breadth of research activity in a focussed subject areas, (11) degree of specialization, visibility of a journal (1) and publication bias. The extent to which each of these factors may have impacted on these findings is uncertain.

These results demonstrate a limited number of published papers on cancer among American Indians and Alaska Natives and suggest that these studies are primarily conducted by a small select group of researchers. Although the graphic presented in figure 1 suggests a recent temporal increase in the numbers of these publications, it is uncertain whether this trend will be sustained in the future.

To a large extent research activity can be considered to follow the availability of external funding. Historically, federal funding specifically targeted to cancer research among American Indians and Alaska Natives has been limited and it has only been during the last several years that targeted support has become available. Studies currently in progress may help to expand the visibility and knowledge base of this research area. Ironically, federal support generally parallels research activity in an area. Unless the visibility of this research area is enhanced, it is uncertain whether expanded support will be available in the future.

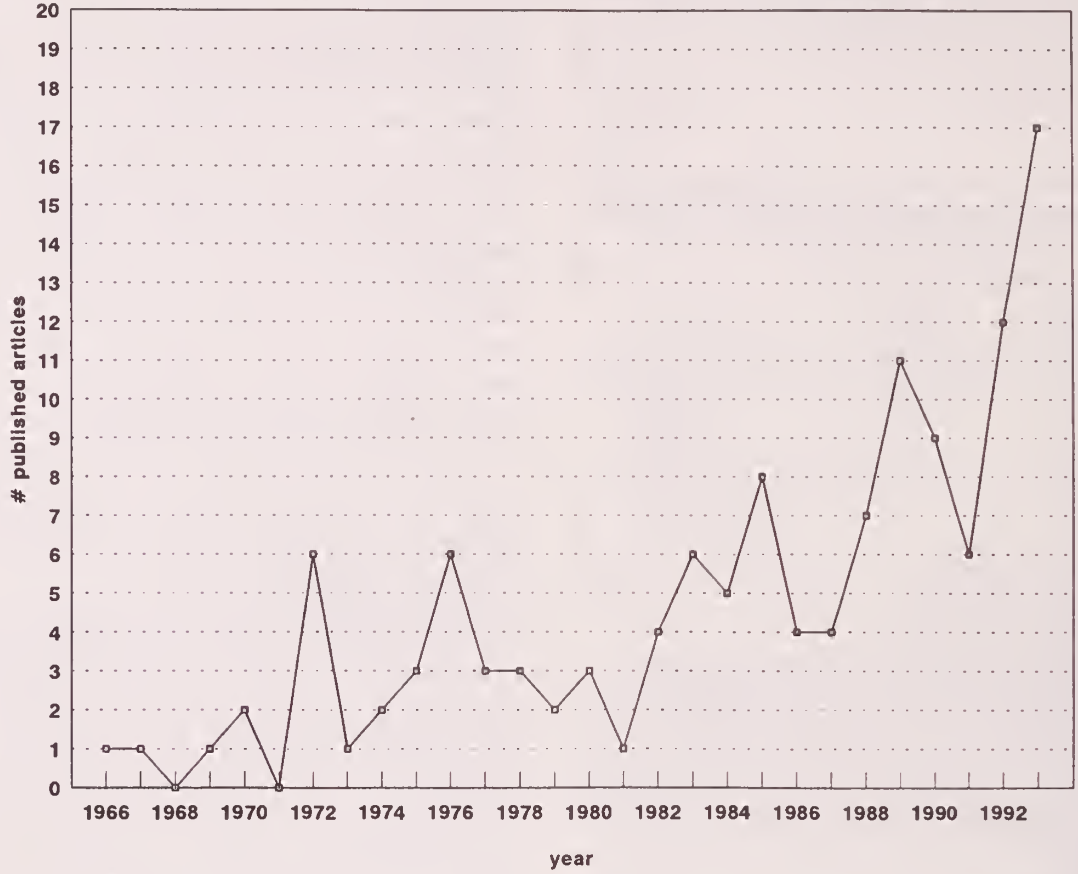
Bibliometric analysis is limited by the fact that it cannot evaluate the content of articles.(12) Nonetheless, a review of titles, abstracts and key word headings suggest that the actual number of population-based studies may represent only a subset of the studies identified in this literature review.

It is apparent that a wide variety of journals have published papers on cancer among American Indians and Alaska Natives serving to complicate broad access to contemporary papers without the assistance of on-line data bases. Additional papers certainly exist in publications not currently indexed on MEDLINE or CANCERLIT (e.g. American Indian Culture and Research Journal, Indian Health Service (IHS) Provider, serial publications, etcetera). A supplemental search of the On-line Computer Library Center (OCLC) data base, (13) which is a national card catalog of all holdings, identified 16 additional records (1966-1993) on this topic including three books, three monographs, proceedings from a conference, seven academic papers (5 theses, 2 dissertations) and two videocassettes; the extent of "peer review" involved in the production of each of these 16 works is probably variable. Also, it is interesting to note that the majority of dissertations have *not* subsequently appeared in peer-reviewed publications.

Although it is difficult to identify all relevant publications, the limited number of identified articles speaks to the focused research activities. This lack of publications may result from limited research support or limited tribal interaction. In addition, tribal groups may experience difficulty in obtaining funding to support research and technical assistance, be reluctant to involve themselves in research, have more immediate concerns which prohibit active involvement and/or may not perceive cancer to be a primary health problem in their community.

While it appears that published cancer studies of American Indians and Alaska Natives are generally limited, this observation may also apply to other ethnic groups. A MEDLINE search of publications from 1976

Figure 1. Number of published articles on cancer among American Indians & Alaska Natives, 1966-1993, by publication year (n=128).



source: MEDLINE & CANCERLIT search.

through January 1995 revealed that only about 2.5% of published studies involving African Americans, Hispanics or American Indians and Alaska Natives focused on cancer. The small proportion of studies on cancer noted among these ethnic groups, is directly reflective of the modest numbers of published cancer studies, that is, both the absolute number and proportion of published cancer studies is limited among each of these groups. Further, these studies appear in a multitude of sources possibly further limiting recognition of the cancer problem among American Indians and Alaska Natives, as well as other ethnic groups.

In an effort to develop a larger perspective in which to assess these results, we used MEDLINE to examine the total number of articles published between 1985 and August 1994 on ten leading causes of death among American Indians and Alaska Natives as identified in the IHS chart series publication for 1993.(14) This search yielded a total of more than 159,000 papers of which only 231 (0.15%) related to American Indians or Alaska Natives. Studies involving American Indians and Alaska Natives represented a comparably small proportion for all health conditions examined. Few papers have been published on causes of death noted to be more common among Native populations (e.g., diabetes, accidents).

There is a sense of urgency to identify and control cancer among American Indians and Alaska Natives. At the turn of the century cancer was unknown among Native peoples. Even thirty years ago cancer among American Indians was rare. Since then there have been marked increases in cancer morbidity and mortality. Cancer education/research among American Indians and Alaska Natives needs to keep pace with these changes. One example of responding to this problem is the Network for Cancer Control Research Among American Indian and Alaska Native Populations developed by the Special Populations Studies Branch of the Division of Cancer Prevention and Control of the National Cancer Institute. Among other activities, this Network has developed a strategic plan (2) to increase awareness of the cancer problem among these unique groups.

It is hoped that this paper will function to increase the awareness of cancer as an important health problem among American Indian and Alaska Native populations and thereby serve to stimulate additional cancer-related research activities involving these groups.

(continued on page 77)

A Bibliography of Cancer Among American Indians & Alaskan Natives, 1966-1994

compiled by Martin C. Mahoney, M.D., Ph.D.(1)
Arthur M. Michalek, Ph.D.(1)

PURPOSE

Cancer represents an increasingly important health problem among American Indian and Alaska Native populations (1-3). Individual research studies of cancer among American Indian & Alaska Native populations have appeared throughout the scientific literature. In addition, two bibliographic compilations included papers on cancer in American Indian and Alaska Native populations.

Justice (1989) (4) prepared a bibliography of cancer in American Indians and Alaska Natives during the period 1800-1989, however the data sources for this work were not described. More recently, Owen et. al. (5) published an extensive bibliography of North American Indians and the health-related scientific literature covering the years 1950-1988. The bibliography by Owen and colleagues had a more generalized focus on AI/AN health issues. In addition, neither of these documents was able to include papers published beyond the late 1980s.

The purpose of this document is to provide a bibliographic summary of cancer among American Indians and Alaska Natives between 1966 and 1994.

METHODS

A MEDLINE (8) search was completed for relevant articles published between 1966 (initial year available on CD-ROM) and 1994. Search terms included: "Indians, North American," "Alaska," "Eskimos," "neoplasms," "cancer" and "cancer control and prevention."

Although MEDLINE does not include all papers on this topic this data base includes citations from an extensive number of high quality, peer-reviewed journals which have each satisfied specific criteria for inclusion in this indexing system. In an effort to maximize the comprehensiveness of this search, the MEDLINE inquiry was supplemented by two additional sources, CANCERLIT and the On-line Computer Library Center data base, using similar search criteria. The CANCERLIT data base included publications appearing between 1983 (initial year) and 1994. The On-line Computer Library Center (OCLC) data base (13) represents a national card catalog data base including books, monographs, academic papers and audiovisual productions.

As noted, only works published between 1966 and 1994 have been included. Non-English citations as well as articles on South American Indians and Greenland Indians were excluded, as were works which appeared to be extraneous and/or have classified American Indians and Alaska Natives with other general population groups.

RESULTS

Searches of the MEDLINE, CANCERLIT and OCLC data bases identified a total of 157 works relevant to cancer among American Indians and Alaska Natives, published between 1966 and 1994. In addition to journal articles, this search was able to identify other works germane to the topic including books, book chapters, conference proceedings, editorials, monographs, theses/dissertations, audiocassettes and serials. The extent of "peer review" involved in the production of each of these works is likely variable.

A bibliometric analysis of studies of cancer morbidity and mortality among American Indians and Alaska Natives was recently completed (9). That paper considered the extent to which this research area has been featured in the scientific literature (1966-1993), sources of information on this topic, the frequency of citations and temporal trends as objective parameters of the breadth and importance of this research area.

DISCUSSION/IMPLICATIONS

It should be acknowledged that additional papers certainly exist in publications not currently indexed on the data bases utilized for this compilation. This includes papers appearing in the American Indian Culture and Research Journal, the Indian Health Service (IHS) Provider and serial publications.

It is apparent that a wide variety of journals have published papers on cancer among American Indians and Alaska Natives serving to complicate comprehensive access to contemporary works.

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(1) Roswell Park Cancer Institute, Buffalo, New York.

This document is intended for use by nascent researchers, "seasoned research veterans" or the intellectually inquisitive. It is hoped that this document will serve to stimulate additional cancer-related research activities involving American Indian and Alaska Native populations as a means of both quantifying and increasing the awareness of cancer as an important health problem, as well as optimizing health status among these groups.

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[end]

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From Out of the Past. . . Over Thirty Years Ago

by Gloria K. Park, M.D.

Vol. 6 No. 3, September 1964, Alaska Ferry Taku near Ketchikan

Dr. Joseph P. Deisher, President of Alaska State Medical Assoc. 1964.

SCIENTIFIC ARTICLES

"Epidemiology of Draining Ears & Hearing Loss in Alaska" by Jacob Brody, M.D. Arctic Health Research Center.

"Alaska 1963 Oral Cancer Survey" by Robert W. Smith, DDS.

"Atherosclerotic Coronary Heart Disease" by Jeremiah Stamler, M.D., Northwestern University Medical School presented at the annual convention, Ketchikan, 5/28/64.

"Sheehan's Syndrome" by F. Jay Keefer, M.D., Bethel, Alaska.

"Cancer Screening Program" by Jim Justice, M.D., Sitka, Alaska, an itinerant field program, Mt. Edgecumbe, USPHS Hospital.

EDITORIALS AND REPORTS

A Congenital Defects Clinic was held for the first time in Alaska under the joint sponsorship of the Anchorage Chapter of the National Foundation, the Alaska Department of Health, and the Alaska Crippled Children's Association. They brought to Anchorage from the University of Washington an outstanding pediatrician, Dr. William Robertson, and a research geneticist, Dr. Fred Hecht, both of whom had had extensive previous experience in the field of congenital malformations.

In the course of two days approximately fifty patients—primarily babies and children with proven or suspected inherited chromosomal, metabolic, and other defects—were presented by private, Native Health Service, and military physicians.

Local physicians were brought up to date on the latest diagnostic tools—and were introduced to newly recognized syndromes. The visiting specialists, on the other hand were introduced to at least one congenital sex-linked bone and joint dysplasia, occurring in Alaskan Eskimos of the lower Kuskokwim region, that to their knowledge had never previously been reported.

The President's Page was devoted to a discussion of professional liability insurance.

The Alaska Department of Health reports that more than 440 active and probably active cases of tuberculosis were found during 1963 in Alaska. Undetermined whether this represents a real increase or reflects refined case detection.

USPHS area-wide conference was held on *"The Development & Implementation of a Village Health Program"*.

Dr. Luther L. Terry, Surgeon General of the USPHS, visited several PHS and military facilities in Alaska. He also spoke at commencement ceremonies of the University of Alaska and received an Honorary Doctor of Laws degree.

ASMA 19TH ANNUAL CONVENTION

May 26-30, 1964 at Ketchikan, registration $5.00, banquet $5.00. International in scope as joined by the British Columbia Association of General Practice.

Arrival at Heckman Wharf of Convention Cruise Ship SS Princess Patricia and 150 doctor couples of General Practice Section of British Columbia Medical Association.

MUKTUK MORSELS

The 7th annual heart clinic was held in Anchorage, Fairbanks and Mt Edgecumbe by a team from the Mayo Clinic.

The 15th annual Alaskan Science conference was held in Fairbanks at the University of Alaska.

Dr. A. B. Colyar spoke on Alaska's experience in tuberculosis control and isoniazid propylaxis at the Pan American Health Organization in Mexico City.

Other Arctic Health Research Center projects have included a rubella outbreak on St. Paul Island and a statewide rubeola vaccine program.

Valdez — Harborview Nursing home which was destroyed during the March 27th earthquake will be reactivated temporarily in the Valdez Community Hospital.

Bethel — Dr. Rober Fortuine formerly SUD (Service Unit Director) at Kanakanak has moved to Bethel as SUD.

Kotzebue — Dr. William Compton has joined the staff.

Glennallen — Dr. Pinneo is sole as Dr. Schneider is on sabbatical.

Kodiak — Dr. Bruce Keers is leaving for a radiology residency.

Anchorage — New physicians include Drs. Ollie and Wally Dunn, Dr. Michael Hein, Dr. Sam DePalatis and Dr. David Ekvall. Also Drs. Robert and Shirley Fraser have returned - he as Tuberculosis Control officer and Shirley at the Alaska Native Service Hospital. Other new physicians are Dr. Alistair Chalmers and Dr. Tryon Wieland who is leaving the Air Force.

Seldovia — Dr. O. H. Armstrong has returned.

Soldotna — A community hospital project has been initiated by Dr. Paul Isaak.

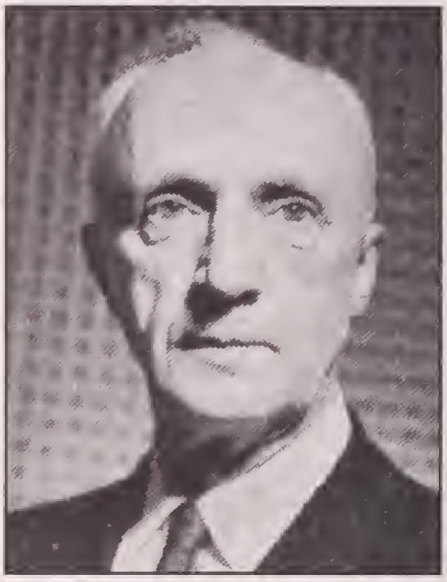
Ketchikan — Dr. Arthur Wilson, Jr. has returned.

Juneau — Dr. Whitehead lost by a narrow margin his bid for re-election to the State House of Representatives.

Homer — Dr. Jack Fenger is active on the Homer City Council.

Several physicians plan to leave the state — Dr. Simpson from Eagle River; Dr. Keers, Kodiak; Dr. Deisher, Seward; Dr. Fenger, Homer; Drs. John and Jean Chapman of Cordova, Dr. Schirmer, Bethel; Dr. Sandburg, Sand Point.

President of the Anchorage Medical Society is Dr. Rodman Wilson, and Dr. Robert Wilkins is President of the Greater Anchorage Health District.



IN MEMORIAM

A. Holmes Johnson, M.D.

Died at Kodiak August 21, 1964, age 69.

Practiced in Kodiak 1938-1960.

Born October 18, 1894 in Iowa.

Graduate of Northwestern University in Chicago and practised in Oregon prior to moving to Alaska.

(continued from page 62 - Bibliometric Analysis)

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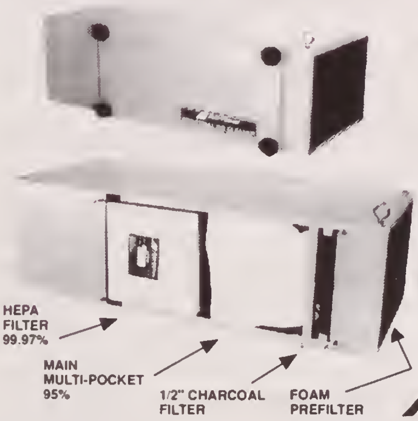

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GLIMPSES OF ALASKAN MEDICAL HISTORY

Edited by Robert Fortuine, M.D.

An epidemic of mumps at New Archangel (1843-44)

Aleksandr Danilovich Romanovskii was the immediate successor to Eduard Blaschke at the hospital at Sitka, or New Archangel. The son of a physician, he received his medical education in Moscow and took service with the Russian-American Company in 1839, arriving in Sitka May 1, 1840, on the same ship which brought the new Governor, A. A. Etholen, and his family, and which took Blaschke home. He remained in the colonies until 1845, when he left by ship for Aian, to return overland through Siberia to European Russia. In 1841 he made a trip to the Aleutians in the company of the governor, during which time he prepared many natural history specimens which he brought back to the capital. He was also said to have visited the outpost at St. Michael.

Several hints suggest that he was a rather difficult employee during his sojourn in Sitka. According to Pierce, Etholen wrote to the main office in 1842 that Romanovskii was denied a bonus and felt he was "abused." Two years later the Sitka office manager complained of the doctor's "rude conduct," which led to an apology to the governor.

Romanovskii, upon his return to Russia, wrote several medical articles about his experiences in the colonies. These were published in German as well as in Russian at St. Petersburg in the later 1840s. These articles contain a wealth of information about medical conditions in Russian America, laced with speculations, popular at the time, relating disease patterns to weather, climate, and geography. The following excerpt describes an epidemic of mumps in Sitka, one of several disease outbreaks which occurred during his sojourn.

"Among the local contagions arising from air changes imperceptible to us must be included parotitis, prevalent in Novo-Arkhangel'sk during December, January and February 1843-1844, which struck all of the natives here without exception, . . .

"This malady spread, like all epidemic diseases here, from SE to NW over an area, so far as we know, of 200 Italian miles. The disease appeared among the Kolosh [Tlingit] living in the southern part of the Straits in October, and continued until the end of November 1843, raging continually with identical force and afflicting people of both sexes and of all ages. . . .

"Parotitis (Cinanche Culleni, Angina parotiquea, mumps, Bauermoetzel) is a fever of the parotid glands, and sometimes also of the submaxillaries, producing swelling and hardness in the stated glands, difficulty in chewing, yawning, and swallowing, and accompanied by severe inflammation.

"This disease falls into two main types: a) flesh parotitis (sarcosa), striking the very substance of the gland and its cellular tissue, and b) nerve parotitis, in which the inflammation strikes the parotid nerve. . . .

"This latter type of parotitis struck the majority of the inhabitants of Novo-Arkhangel'sk during the three months noted. However, during an epidemic, flesh parotitis frequently appeared here also combined with nerve spasms. The following phenomena accompanied this illness: for a day or two before its appearance the patient felt shooting pains in one or both partotid glands; then a small swelling appeared in them, without surface redness; toward evening very slight feverish movements were displayed (somethimes they did not appear at all) light headache and loss of appetite; other functions remained normal. Swelling continued several days, with body heat, particularly in the afflicted parts. With normal rubbing of volatile ointment, the disease ran its course without any aftereffects. These were the lightest cases. . . . The immediate cause of the disease lay in the affliction of the parotid nerve, and its appearance depended solely on a miasmatic condition of the atmosphere, mainly characteristic of the autumn and winter seasons. In extremely rare cases the illness transferred to the breasts and generative parts in women, and to the testes in males. The course of the illness was brief; in light cases, it did not last more than four or five days; in more severe cases it lasted not less than ten or twelve. . . .

"Some contend that parotitis may be infectious but with the epidemics we had this was not the case. The disease, as already stated, acted only on the natives: Creoles, Aleuts, and Kolosh; Europeans were not subject to it at all, in spite of close relations with them and similar conditions.

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Letter to the Editor

MEDICINE AS A CALLING

Dear Editor,

James S. Todd, the AMA executive vice-president said recently, to date, no one has proved any one best way to deliver health care, and we have become a little bit tired of zealotry on both sides of the aisle." (*American Medical News*, March 27, 1995)

Somehow Todd has missed the point, and his stewardship is warped in quite the wrong direction. In our opinion, the best way to practice medicine would be to have all physicians educated at government expense, and to have all doctors live permanently in hospitals, available at all times, and provided with room, board, laundry and Prozac. Unfortunately, medicine is still not an absolute calling; it is still a profession - although day-by-day it becomes closer to a job. Todd and the AMA have been actively failing to recognize that the AMA was, before Morris Fishbein, an organization of professionals, and one of its purposes was to promote the professional well-being and independence of the physician. Fishbein changed all that; Sammons followed suit, and Todd could care less — as long as he has job security.

There have always been physicians who preferred to be salaried, and that's fine, as long as there is a viable option to it. The growth of HMOs and other managed "care" groups, however, is reaching monopolistic proportions, and those organizations have no dedication to physicians' well-being, or even the well-being of clients, as long as the latter receive sufficiently adequate service to continue buying the package. I am sorry if this zealotry on this side of the [a]isle is offensive to Todd, but he should be actively opposing the industrialization of medicine, not playing footsie with the GHAA and the HCFA.

George C. Manning, M.D.
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PREVALENCE OF ALCOHOL AND ILLICIT DRUG USE BY EXPECTANT MOTHERS

Jack Jacob, M.D. [(1)]
Harry Harrison, Jr., M.D. [(1)]
Anita Todd Tigert, M.P.H., M.S.N. [(1)]

ABSTRACT

Background: **Prevalence data of illicit drug and alcohol use during pregnancy in Alaska is limited. This study demonstrates the prevalence of perinatal drug and alcohol use at a single community hospital in Anchorage, Alaska.**

Methods: **A convenience sample was used during a six month period. 351 anonymous urine samples were obtained from imminently delivering women. Screening toxicology tests were run and results were linked to demographic variables using a common random reference number.**

Results: **16.2% (n=57) of the samples were positive for drugs or alcohol. Cannabinoids and alcohol were found most frequently. Polydrug use was found in 22.8% of the positive samples. Medicaid recipients represented the highest users of illicit drugs and alcohol.**

Conclusions: **The prevalence of positive drug screens and polydrug use in our sample is amongst the highest reported in the literature. The high prevalence of drug and alcohol use indicates that present prevention strategies are not working in Alaska. New and innovative outcome based strategies to decrease drug and alcohol use in pregnancy are needed.**

BACKGROUND

Drug use during pregnancy has been associated with many adverse outcomes including spontaneous abortions, intrauterine growth retardation, and birth defects. Campaigns to target drug use by pregnant women have been poorly received by health care providers. Reasons for this include lack of available resources in the community, provider discomfort with eliciting drug use information, and lack of acknowledgement by practitioners that drug use occurs in their patients. Prevalence data is necessary in order to determine if there is a community need before funds can be allocated to support prevention and rehabilitation programs for pregnant women. Nevertheless there has been little information on the prevalence of drug use in Alaska during pregnancy.

Using urine drug screening to determine prevalence remains controversial. Patient confidentiality and legal issues surrounding drug screening has limited the number of studies being conducted. This is especially true for perinatal drug use where concerns for both the mother and the infant must be confronted (1). However, unless policy makers are willing to allocate funding for services based on perceptions of providers, universal anonymous screening is the only objective method to obtain prevalence data.

We had originally planned to study the prevalence of drug use among all delivering women in Anchorage, Alaska. Only one of four hospitals in Anchorage would approve the study based on its anonymous screening design. The purpose of this study, therefore, is to determine the prevalence of drug use among women delivering at a single community hospital in Anchorage, Alaska.

METHODS

Using a convenience sample from a local community hospital, anonymous universal screening of imminently delivering women was conducted for a six month period, June 15, 1993 through December 14, 1993. The study design was approved by the Institutional Review Board of the hospital. Procedures were established and followed to ensure that no identifying information could be linked to the urine samples. Demographic and sample results were linked by a computer generated random reference number that was assigned at the time of specimen collection. Descriptive demographic information (age, race, payor status, and urban or rural residence) was collected by the labor and delivery nurses. Payor category was used as a proxy for socioeconomic status. This information was separated from the urine sample prior to sample analysis. Identifying information was omitted to protect patient confidentiality. A urine sample was collected by the floor nurses as soon after admission to the hospital as the patient was able to void. Samples of patients given medications for pain management or cesarean section prior to specimen collection were identified by the nurse. The drug given and time from sample collection were recorded. Specimens testing positive under this condition were excluded from analysis. Urine samples were collected in batches and run at the end of each month, thus eliminating any deductive identification of results. Samples were analyzed at the PharmChem Laboratory in Menlo Park, California using the EMIT (enzyme-multiplied immunoassay techniques) and Syva reagents and enzymatic assay for alcohol. Confirmation testing was not done to eliminate the possibility of utilizing positive findings for criminal prosecution. Table 1 lists the drugs screened, methods used, cutoff levels, and detection periods for each drug.

---

(1) Alaska Neonatal/Perinatal Research Foundation, Inc., 3340 Providence Drive, Suite 366, Anchorage, Alaska 99508 and Alaska Science & Technology Grant #89-1-199.

Table 1.

Drug Testing Panel

Drug Class	Screening Method	Screening Levels	Detection Period**
Amphetamines	EMIT	1000 ng/ml	12-72 hours
Barbiturates	EMIT	200 ng/ml	2-4 days*
Benzodiazepines	EMIT	200 ng/ml	Up to 30 days
Cannabinoids	EMIT	20 ng/ml	Up to 14 days
Methadone	EMIT	300 ng/ml	1-4 days
Opiates	EMIT	300 ng/ml	2-4 days
Phencyclidine	EMIT	25 ng/ml	Up to 14 days
Cocaine metabolite	EMIT	300 ng/ml	12-72 hours
Alcohol	EA	0.02%	very short

*Phenobarbital may be detected up to 30 days.
**(Vega, Kolody, Hwang, & Noble, 1993)
EMIT = enzyme multipled immunoassay technique
(error margin ± 1.5%)
EA = enzymatic assay

Descriptive statistics were used to analyze the demographic information. Chi-square and Fisher's exact test were used to compare positive samples to the nonuser population. Sample size determination was based on 15% of women using drugs prior to delivering (using prevalence data (2) obtained in 1990 from another major city in the state). An estimate of 310 samples were necessary to obtain a 95% confidence limit fixed at ± .04.

RESULTS

Three hundred seventy-two samples were initially obtained. Seven positive screens were removed from the sample set because these screens showed positive for legal drugs used during delivery. Fourteen samples had insufficient quantity to screen, leaving 351 in the drug prevalence analysis. Fifty-seven samples (16.2%) were positive for illicit substances and alcohol. Individual drugs identified are listed in Table 2 by frequency. Cannabinoids (5.4%), barbiturates (4.8%), and alcohol (4.6%) were the most frequent drugs detected.

Thirteen samples tested positive for more than one drug. This represented 22.8% of positive samples indicating a relatively high incidence of multiple drug use. Multiple drug use was represented equally in the 18 -24 (22%) and 25 - 34 (21%) age groups who were positive compared to 38% in the >34 age group. Sixty-nine percent (n=9) of multiple drug users were Medicaid compared to 15% for private payor (n=2). Cannabinoid

Table 2.

Drugs Identified in Pregnant Women at Delivery

Drug Identified	# of Positive Samples *57/351	% of Study Population* (16.2%)	# of Positive Samples† 58/400	% of Study Population† (14.5%)
	Anchorage (1990)		Fairbanks (1990) (2)	
Cannabinoids	19	5.4	40	9.8
Barbiturates	17	4.8	13	3.2
Alcohol	16	4.6	not tested	not tested
Opiates	9	2.6	2	0.5
Cocaine Metabolite	7	2	10	2.4
Amphetamines	6	1.7	1	0.2
Benzodiazepines	3	0.85	not tested	not tested
Phencyclidine	1	0.28	not tested	not tested

* Thirteen women tested positive for more than one drug
† Eight women tested positive for more than one drug

Table 3.

Anchorage and Fairbanks Comparison

	Anchorage 1993* (n = 351)		Fairbanks 1989 (2) (n = 400)	
Demographic Variables	# Positive Sample Size	Percentage Positive (16.2%)	# Positive Sample Size	Percentage Positive (14.5%)
Age: <18 yrs.	0/6	0%	0/17	0%
18-24 yrs.	18/89	20.2%	19/136	14.0%
24-34 yrs.	29/185	15.7%	35/203	17.2%
>34 yrs.	8/53	15.1%	4/44	9.1%
N/A†	2/18	11.1%	**	**
Payor: Self	3/18	16.7%	3/27	11.1%
Private	20/192	10.4%	25/184	13.6%
Medicaid	27/92	29.3%	17/96	17.7%
CHAMPUS	0/2	0%	1/14	7.1%
N/A†	7/47	14.8%	**	**
IHS	**	**	12/78	15.4%
Race: White	44/280	15.7%	35/269	13.0%
Black	3/16	18.8%	4/24	16.7%
Native	2/8	25.0%	18/98	18.4%
Other, N/A†	8/47	17.0%	1/9	11.1%
Location: City	49/262	18.7%	45/326	13.8%
Other	1/34	2.9%	11/69	15.9%
N/A†	7/55	12.7%	**	**

* barbiturates and opiates included except where prescribed by physician as indicated on the record

** data variable not collected

† information not available during data collection

use was indicated in 50% of cases of multiple drug use. Amphetamine, barbiturates, cocaine, alcohol, benzodiazepines and PCP were all identified in drugs of polyuse.

Table 3 shows prevalence of positive samples related to certain demographic variables. It also includes comparison data for a similar drug prevalence study from Fairbanks, Alaska reported in 1991 (2). There was a significant relationship between payor status and a positive drug screen (P = .001). Women on Medicaid were 2.8 times more likely than women with private insurance to have a positive drug screen. Of the positive screens, cannabinoids (P = .01) and cocaine (P = .001) were more likely to be used in the Medicaid population than in the private or self-pay group. Viewed in another way, although Medicaid recipients represented 29% of the population they accounted for 67% of the non-alcohol positive screens, 86% of positive cocaine screens, 58% of positive marijuana screens and 67% of positive amphetamine screens (See Figure 1). On the other hand, the above drugs were underrepresented in the non-Medicaid population (see Figure 2). Alcohol on the other hand, was evenly distributed among private and Medicaid payor categories.

The relationship between age or race to a positive drug screen was not significant (age - P = 0.618 and race - P = 0.958). This finding is consistent with other literature (3,4).

DISCUSSION

Urine screening provides information only for a window of time surrounding the sample collection. It does not provide a picture of what a mother may have ingested during the course of her pregnancy. In addition urine screening provides a much shorter window of detection compared to meconium testing (5). Due to these limitations, it is generally felt that single sample urine specimens underestimate the prevalence of drug use during pregnancy (5,1,6).

Urine alcohol levels do not correlate with blood alcohol levels or the motor performance of the individual who has consumed alcohol. Detection of alcohol in the urine is constrained by the influences of body weight, amount ingested, and time elapsed prior to testing. Alcohol is rapidly absorbed into the blood stream and distributed throughout body water. It is estimated that an average sized person would need to consume 12 oz. of beer, 4 oz. of wine, or 1 oz of distilled spirits to have a positve urine screen (at our cutoff levels) at 1 1/2 hours after ingestion (5). Thus it is apparent that in order to test positive in the urine, alcohol needs to be consumed in fairly sizeable quantity shortly before sample collection. These limitations also make it likely that urine screening underestimates the true incidence of alcohol use during pregnancy. The implications of alcohol use late in pregnancy is also different than that of other drugs. Much of the teratogenic effects of alcohol have been documented with high alcohol intake early in pregnancy. Despite these limitations we felt that information regarding alcohol use just prior to delivery would provide some basic prevalence information. This is especially true

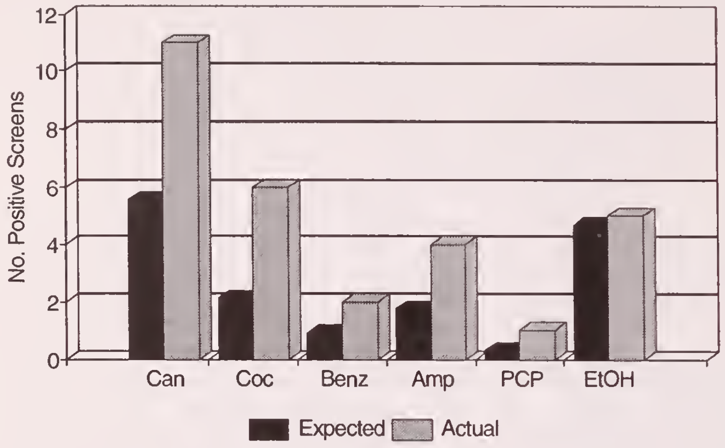

Figure 1:Expected vs. Actual Positive
Medicaid Drug Screens

for Alaska where alcohol consumption is a major health problem and little prevalence information is available on alcohol use during pregnancy.

Another limitation of our study is that it does not give population based information. It was our original intent to study drug prevalence in all four hospitals in Anchorage. However, Institutional Review Board and administrator approval was granted in only one of the four hospitals. The major obstacle in obtaining study approval was our study design calling for specimen testing without patient consent inspite of the fact that our study design has been used nationally for obtaining prevalence data and included safeguards for preventing identification of individuals. Obtaining consent prior to drug testing has major limitations since it may exclude the very portion of the population most likely to have a positive screen. Information from such a consent-based study design can no longer therefore be considered a prevalence study. Our experience underscores the difficulty in undertaking a study of this vital public health problem in Alaska.

This survey and a similar one done in Fairbanks reported in 1990 (2), demonstrated that approximately 14-16% of maternity patients had used an illicit drug or alcohol within hours or days of delivering. There were several differences between the studies: 1) the Fairbanks study was done four years earlier; 2) alcohol was included only in our study; 3) there was greater Indian Health Service and Native representation in the Fairbanks study; and 4) personal possession of small quantities of marijuana was legal in Alaska when the Fairbanks study was conducted. Nevertheless, both studies demonstrate a remarkably similar incidence of positive drug screens (16.2% for Anchorage and 14.5% for Fairbanks). These numbers are similar to those reported in Florida (14.8%) (7) but higher than those reported in California (11.4%) (5) and Utah (7.8%) (3) and in a midwest city (9.5%) (8).

Cannabinoids were the most common drug testing positive in both the Fairbanks and Anchorage study. The use in Fairbanks was much higher (9.8% versus 5.4%) and may be a result of the fact that possession of marijuana was legal in Alaska in 1990, a situation which has since changed. Alcohol was the third most common drug detected (4.6% of samples). Alaska's Pregnancy Risk Assessment Monitoring Survey (PRAMS) found that in 1990-1991 10% of women responding to this self-report survey used alcohol during the last trimester of their pregnancy (9). This is surprising despite the public information that is available in Alaska on the adverse effects of alcohol use during pregnancy. This is also useful information for health care providers who care for newborns who frequently consider illicit drug use but not alcohol use when faced with an infant who is demonstrating poor arousal and increased irritability.

Polydrug use was found in 22.8% of our positive samples. Only two women in this group were positive

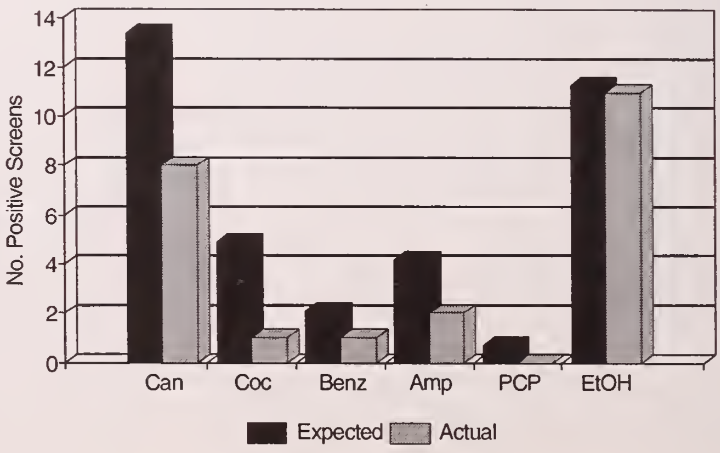

Figure 2:Expected vs. Actual Positive
Non-medicaid Drug Screens

for alcohol plus another drug. These two women were also positive for cannabinoids. Marijuana and alcohol are common partners in polydrug use (3). The level of polydrug use in the Anchorage sample is concerning since most authors report a prevalence between 0.5% and 4% (10,5,11). The Pinellas County study reported polydrug use of 11% (7).

Our study demonstrates that users of Medicaid have a greater proportion of positive drug screens, especially illicit drugs. This finding is contrary to the Pinellas County study (7) that found the prevalence of illicit drug and alcohol use was similar in women receiving care in both a public and private care setting. However, our finding of a higher prevalence in Medicaid mothers has been reported elsewhere (3,10,8,4). On the other hand, age-specific information indicates that women of all ages are involved in drug and alcohol use during pregnancy. Unfortunately, there was not enough diversity in population (race and location variables) to draw any conclusions. These remain areas for further study.

Another area for further study is the issue of alcohol use during pregnancy. It is particularly difficult to get information on this issue based on the limitations of urine alcohol testing. Furthermore, meconium testing, ideal for the identification of long term drug use during pregnancy, is not appropriate for identification of alcohol. This leaves repeated random blood/urine testing of women during prenatal care and delivery as the only testing method to determine alcohol use during pregnancy. Interview techniques described by Hindliter and Zelenak (12) demonstrate that strategies can be incorporated into prenatal history taking during initial and subsequent prenatal visits that improve identification of women using illicit drugs and alcohol. Despite difficulties collecting such data, Alaska is a particularly appropriate state for conducting a study to determine a more accurate prevalence of alcohol use during pregnancy.

In summary, our study demonstrates a relatively high incidence of illicit drug and alcohol use in pregnant women prior to delivery. Furthermore, we have identified a certain category of patients (Medicaid recipients) who are at highest risk for illicit drug use. This information should be useful for health care planners and perinatal care providers. The high prevalence of drug and alcohol use indicates that present prevention strategies are not working in Alaska. New and innovative outcome-based strategies to decrease drug and alcohol use during pregnancy are needed. Finally, this information also underscores the potential health care costs related to high risk social behaviors during pregnancy.

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Collaborative Visual Rehabilitation:

HIGH ASTIGMATISM, ESOTROPIA AND ELEVATOR PALSY

Robert W. Arnold, M.D.(1)
Lynn Coon, O.D.(2)
Patrick Reber, O.D.(3)
M. Diane Armitage, C.O.(1)

INTRODUCTION

Approximately 5% of children are subject to the potential ravages of the three primary types of amblyopia: strabismic, deprivational and refractive. Refractive amblyopia is commonly associated with unequal hyperopia (anisometropia) but can occur with uncorrected astigmatism. Astigmatism is usually due to a non-spherical cornea shaped (like the side of a football while spherical optical surfaces resemble a basketball. The ectatic corneal dystrophy, keratoconus resembles the point of a football.) If amblyopia is not successfully treated before age ten and the resultant visual acuity is less than 20/40, then driving will be restricted. Vision less than 20/70 qualifies for "impairment" and less than 20/200 will be "legally blind."

We report a case exhibiting atypical and severe risk factors for strabismic and refractive amblyopia for which visual rehabilitation required a collaborative effort of orthoptist, optometrists and pediatric ophthalmologist.

CASE REPORT

A thirteen-year-old male was referred for poor vision, high astigmatism and strabismus. Half correction of anisometropic astigmatism yielded 20/200 vision in the right eye and 20/50- vision in the left eye with a small angle hyperdeviation.

The patient was first seen by James H. Patterson, M.D. at age 15 months for "crossed-eyes since birth" despite a negative family history for strabismus or amblyopia. He demonstrated a 35pd esotropia with left fixation preference. An atropine cycloplegic refraction yielded +2.50 + 0.75 x 90 o.u. The remainder of his exam was normal and specifically excluded ptosis, anisometropia or incomitance. After one month of occlusion therapy, the fixation preference was eliminated and the near deviation measured 40pd. Therefore, he underwent bilateral medial rectus recession of 5mm via a limbal approach. One week and one month post-operatively he remained orthophoric for distance and near targets without spectacle correction.

At age 13, his visual acuity corrected to 20/60 with -5.25 + 9.00 x 100 O.D. and 20/20- with +0.25 + 4.00 x 88 O.S. Enhanced Brückner Test (1) was abnormal with a horizontal light source (figures 1,2). Fixation with a right hypodeviation (left hyperdeviation) was inconsistent due to poor uncorrected acuity. The patient had an unremarkable anterior segment and retinal exam other than a mild right ptosis: vertical fissures 6mm O.D. and 7mm O.S. with 15 mm excursions O.U. Keratometry readings were 46.75 x 42.00 @ 04° O.D. and 45.75 x 42.50 @ 04° O.S. The patient then underwent a careful optometric fitting of daily-wear, gas-permeable contact lenses in each eye. The left eye was fit with a spherical, Boston IV®, 7.76 mm Base curve, 9.3 mm diameter with power of +1/50 diopters. The right required a bitoric Boston IV® with base curve of 7.89 x 7.11 mm, diameter 9.5 mm and back vertex power of +2.50 -6.50 x 180°. The Enhanced Brückner Test was significantly normalized (figures 3,4) and the patient's best corrected acuity was 20/40- O.D. and 20/20 O.S!

Corneal Topography revealed anisometropic, steep regular astigmatism without classic characteristics of an ectatic corneal dystrophy (figure 5). The 14pd left hyperdeviation increased to 18pd in right gaze and to 20pd in right tilt with the remainder of the Bielschowsky Head-Tilt test consistent with a mild right superior rectus palsy confirmed by Hess Test (figure 6).

DISCUSSION

This case demonstrates the advantage of combined-modality treatment of potentially disabling multidimensional eye disease. Initially presenting as

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(1) Pediatric Ophthalmology and Strabismus, Ophthalmic Associates, a P.C., 542 West Second Avenue, Anchorage, Alaska 99501-2242.

(2) Ophthalmic Associates, 935 East Westpoint, Wasilla, Alaska 99645.

(3) Alaska Eye Care Centers, 1345 West 9th Avenue, Anchorage, Alaska 99501.

Correspond and reprint requests: Dr. Arnold, 542 West Second Avenue, Anchorage, AK 99501-2242.

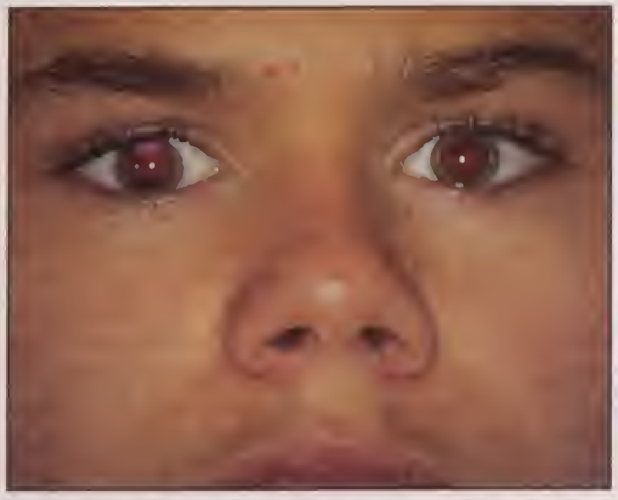

Figure 1. Enhanced Brückner Test horizontal light source of patient without correction. (Olympus OM-2SP, Tokina 500mm Mirror, T-32 flash, Ektachrome 100). Vision 20/300 od, 20/70 os.

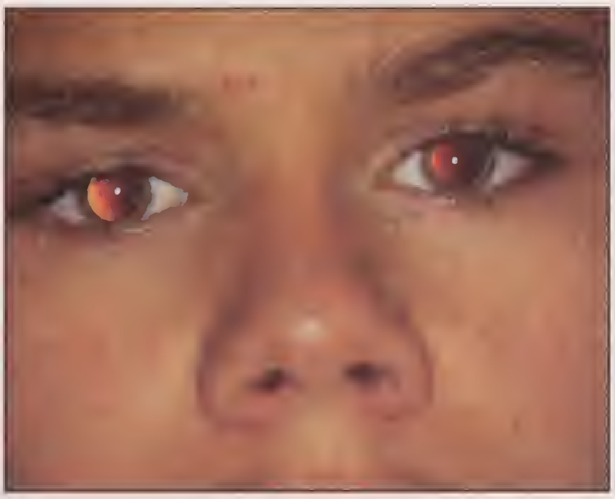

Figure 2. Enhanced Brückner Test vertical light source of patient without correction. Cycloplegic refraction: -5.25 + 9.00 x 100 O.D. and +0.25 + 4.00 x 88 O.S.

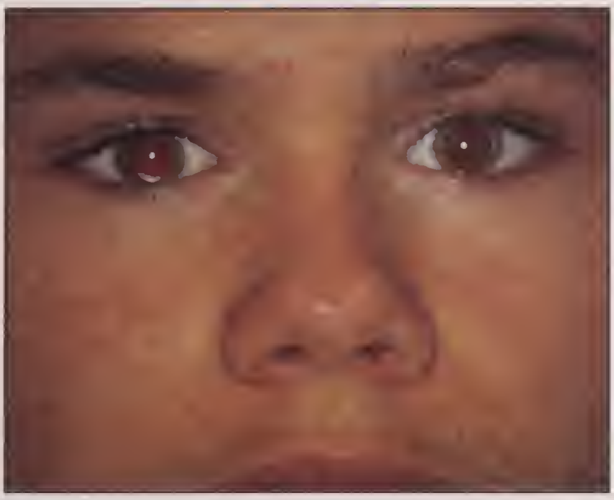

Figure 3. Enhanced Brückner Test horizontal light source of patient corrected with bitoric rigid, gas-permeable contact lens, right eye and a spherical, gas-permeable contact lens, left eye. Vision 2040- right eye and 20/20 left eye.

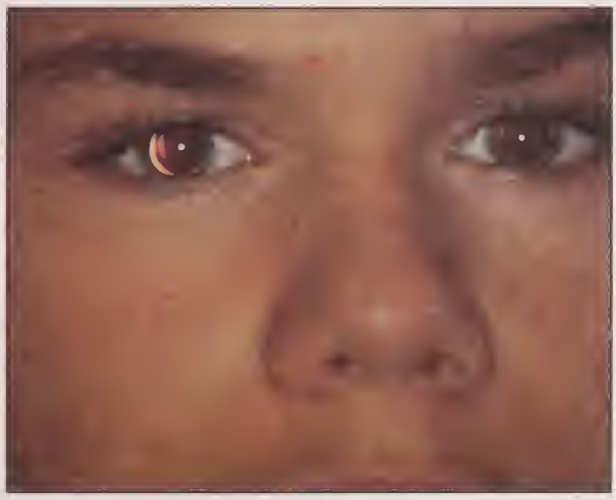

Figure 4. Enhanced Brückner Test vertical light source of patient corrected with a bitoric, gas-permeable contact lens, right eye and a spherical, gas-permeable contact lens, left eye.

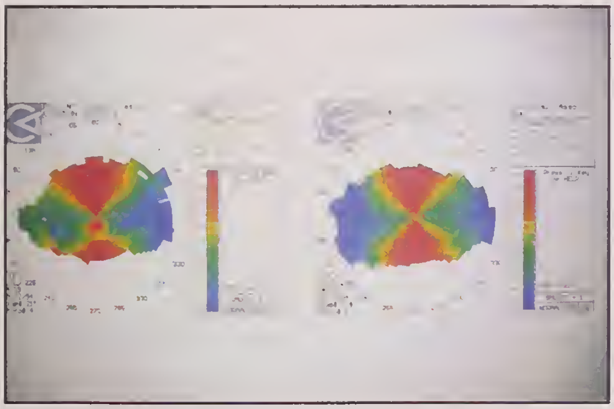

Figure 5. Corneal Topograph of right and left eyes (oriented with right eye on left). Red indicates steep corneal curvature of 47.8 diopters while blue indicates flat curvature of 39.8 diopters right eye and 45.3 diopters while blue indicates flat curvature of 41.2 diopters, left eye. (Tomey Instruments)

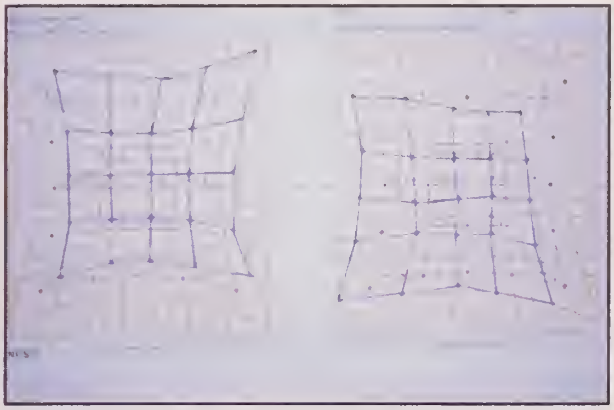

Figure 6. Hess Test of Comitance indicating right superior rectus palsy.

typical essential infantile esotropia without significant refractive error, the patient responded as expected to surgical intervention. Then a mild elevator palsy intervened to produce a hyperdeviation and mild ptosis. These factors probably combined to produce anisometropic, severe acquired with-the-rule astigmatism and partial amblyopia of the right eye. Ptosis and vertical strabismus are known causes of astigmatism but rarely to this degree(2). His rehabilitation required highly specialized fit of a bitoric hard contact lens and collaborative strabismus assessment and treatment. With prudent contact lens wear, this student can now function with "normal" vision and without the fear of blindness should he lose his best eye. The potential progression of astigmatism can be accurately followed by corneal topography but will probably be retarded by contact lens wear. The hyperdeviation will be treated by superior rectus muscle resection.

This case also emphasizes an inherent limitation on photorefractive visual screening (3). Even in the presence of large, amblyogenic astigmatism, the photograph of the Enhanced Brückner Test and photoscreeners designed to detect mild-to-moderate refractive errors (i.e., M.T.I.) may be relatively unimpressive (Figure 1,2, left eye) when the astigmatism is mixed (combined hyperopic and myopic).

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OCULAR COMPLICATION IN HIV INFECTED INDIVIDUALS

Thomas J. Harrison, M.D.(1)

ABSTRACT

The incidence of acquired immunodeficiency syndrome is growing within all levels of Alaskan society. All physicians will be touched in some manner by this epidemic and there is a growing need for a thorough understanding of all aspects of the human immunodeficiency virus and acquired immunodeficiency syndrome. Visual complications associated with this disease are devastating, common, and in many cases treatable. The following discussion is an overview, in outline form, of the ocular complications associated with the human immunodeficiency virus and acquired immunodeficiency syndrome.

The magnitude of human immunodeficiency virus (HIV) infection and resultant acquired immunodeficiency syndrome (AIDS) is staggering. A conservative estimate is that by the year 2000, thirty million adults and 10 million children will be infected by the human immunodeficiency virus (HIV). In the United States 243,423 deaths due to AIDS have occurred through June, 1994 and about 40,000 new cases are diagnosed annually (1). Further with no definitive treatment or vaccination the epidemic is still in it's infancy. AIDS affects all body systems and no physician will be free from the responsibility to be knowledgeable about the manifestations of HIV infection and AIDS.

Alaska with it's young, sexually active population along with abuse of alcohol and drugs is a rich environment for sexually transmitted diseases. It is not surprising that Alaska has not remained immune to the AIDS epidemic. According to state statistics Alaska (Jan. 1995) has had 272 documented cases of AIDS with 152 deaths (2). Out of 75,826 individuals tested for HIV infection, 540 (0.7%) were positive. This number does not include those HIV positive individuals who remain unaware of their disease and those cases unreported. It takes little stretch of the imagination to assume that there is a large reservoir of HIV infected individuals in Alaska who will be seeking care in the next decade.

Visual loss is one of the most common complications of AIDS and certainly one of the most devastating. Timely recognition of HIV and AIDS ocular complications is often difficult, but of extreme importance because many of these vision threatening conditions are treatable. In addition recognition of ocular disease may lead to diagnosis of treatable systemic disease. The purpose of this article is to provide physicians with an overview of ocular manifestations of HIV infection based on a review of current literature, discussions with national AIDS consultants and the personal clinical experience of the author.

Ocular manifestation of AIDS can be divided into four categories: an ocular reaction to HIV known as **non specific HIV ocular disease** or HIV ocular microangiopathic syndrome (OMS); ocular disease produced by **opportunistic infections; indirect** ocular manifestations of systemic disease; and **ocular toxic reactions** to medications used in the treatment of AIDS.

I. NON SPECIFIC HIV OCULAR DISEASE

A. Ocular microangiopathic syndrome (OMS)

Non specific HIV retinopathy or ocular microangiopathic syndrome occurs at an earlier stage of HIV infection than opportunistic infections or indirect ocular disease. Microaneurysms; blot and flame hemorrhages, vascular congestion, and cytoid bodies make up the ocular microangiopathic syndrome (OMS) (3,4,5,6,7,8) Photo #1. Cytoid bodies are small, white to yellow, opaque, inner retinal nerve fiber infarcts within the ganglion cell layer.

OMS is associated with systemic microvasculopathy and lowered helper and inducer T cell levels (CD4). The critical CD4 count for occurrence of retinal cytoid bodies, the main feature of OMS, is a mean CD4 count of 175 cells per cubic mm (7). Usually OMS is asymptomatic, but occasionally can cause patients to experience scotomata (faint blank spots in the visual field that do not move or float. Some studies suggest an association of OMS with cerebral hypoperfusion and cognitive deficits typical of aids dementia (8,9). Subtle visual dysfunction can occur early in HIV infection prior to retinitis. Studies have shown color-vision and contrast-sensitivity defects in patients without non-specific retinitis (10). In addition, major opportunistic infections develop within 3.8 (+/- 3.5) months after appearance of OMS (11).

No treatment is required for nonspecific OMS. However, it must be remembered that similar retinal

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(1) 3500 La Touche, Suite 250, Anchorage, Alaska 99508

changes can occur in treatable diseases unrelated to AIDS such as diabetes, hypertension, radiation retinopathy and others. A helpful axiom is that just because an individual is HIV positive does not necessarily mean that other disease entities, unrelated to HIV, cannot occur. In fact some have observed that diabetic retinopathy and other metabolic diseases in HIV infected individuals may be more severe and require more aggressive management (12).

B. Optic nerve atrophy

Pale optic nerves (optic atrophy) may occur early in HIV infection as a nonspecific ocular complication. HIV appears to be neurotropic by direct viral involvement of glial cells and perhaps neurons by entering the nervous system via infected macrophages, or by damaging the endothelial cells of the nervous system allowing the virus to enter by way of a defective brain/blood barrier. HIV can be isolated from the nervous system within 15 days of infection (13). Tenhula and others counted optic nerve axons of AIDS patients and found that there was a substantial dropout of axons (up to 40%) compared to matched controls (14). This optic nerve degeneration was not associated with opportunistic infection by cytomegalovirus (CMV) or cryptococcus, but apparently was nonspecific related to the neurotropic nature of HIV. There is no treatment for HIV optic atrophy, but caution must be observed not to mistake nonspecific optic atrophy from optic atrophy caused by opportunistic infections or intracrainal lesions.

C. Vitreous cells

Vitreous cells (best appreciated by slit lamp examination) are seen early in HIV infection and can be non specific. They have been noted with high CD4 counts some months prior to CMV retinitis. Vitreous cells can produce symptoms of visual floaters best seen on bright days or against a white background. These should not be confused with age related vitreous floaters which are common in the normal population. Age related vitreous floaters are usually strings of collogen and are rarely cellular in nature. However, when vitreous cells are present, accompanied by low CD4 counts, ocular opportunistic infections must be considered and careful retinal examination is mandatory.

D. Uveitis

Uveitis is an ubiquitous common inflammatory ocular disease manifest by an out-pouring of protein and inflammatory cells into the aqueous and vitreous. Uveitis is associated with many systemic diseases including HIV infection. Uveitis can occur as a primary non specific response to HIV. AIDS associated uveitis due to opportunistic infection and AIDS medications will be discussed later.

E. Keratitis sicca

Keratitis sicca or dry eyes is a commonly overlooked nonspecific disease experienced by AIDS patients. Studies suggest that 12% to 21% of HIV study groups have signs and symptoms consistent with keratconjunctivitis sicca (15). Some authors have associated HIV with a Sjogrens-like syndrome (16,17). Dry eyes are particularly distressing during periods of low environmental humidity such as Alaskan winters. Sympathomimetic antidepressants (for dementia) and methodone (for recovering drug addicts) may exacerbate dry eyes. Lack of adequate ocular moisture may contribute to opportunistic infectious keratoconjunctivitis and intolerance to contact lenses. HIV infected individuals should be offered aggressive therapy for dry eyes. Contact lenses, especially continuous wear contact lenses, should be discouraged.

F. Premature presbyopia

Early onset presbyopia (decreased near vision with age) is an additional nonspecific ocular complication of AIDS. Impaired accommodation was found in 60% of AIDS patients younger that 40 years of age (18). Simple "store bought" reading glasses may benefit these patients.

II. OPPORTUNISTIC OCULAR INFECTIONS

In general three important facts should be emphasized concerning opportunistic ocular infections in HIV infected individuals. The first is that disease manifestations of specific organisms in immunodeficient patients can be quite different from those same entities in immunocompetent individuals. Second is that a specific disease may have several widely different presentations in an immunodeficient individual. Lastly, more than one opportunistic ocular infection can occur simultaneously.

A. Posterior segment

1. Cytomegalovirus (CMV) retinitis

CMV and other ocular manifestations of opportunistic infections begin to occur occasionally with CD4 counts below 100 and regularly with CD4 counts below 50. By far, the most common opportunistic ocular infection is cytomegalovirus retinitis. Twenty five percent or more of AIDS patients will eventually have CMV retinitis (4,5,6,19,20,21). CMV retinitis can be asymptomatic initally or associated with multiple small floaters and blurred vision usually limited to one eye. Vitreous cells can be present, but the eye is usually quiet without redness or photophobia. This is in contrast to other opportunistic infections such as toxoplasmosis and herpes zoster where profound visual symptoms occur early with severe panuveitis.

CMV retinal lesions are irregular white, thick, dry appearing and involve the inner retina (Photo #2). They can occur in the peripheral or central retina. Hemorrhage is a major component of CMV retinitis. Central lesions usually appear along the major retinal vessel arcades, but CMV can involve the fovea and/ or the optic nerve as well (22,23,24). Care must be taken not to ignore the peripheral retina where CMV retinitis can occur initially as a subtle granular change in the retinal pigment epithelium prior to hemorragic CMV retinitis. At times the vasculitis is so severe that the vessels take on a "hoar frost" appearance. This is known appropriately as frosted angiitis (25) (Photo #3).

Systemic CMV involvement of the esophagus, colon, lung, liver and brain frequently accompany eye disease. Appearance of these systemic signs and symptoms should raise suspicions of eye involvement. Recognition of CMV retinitis is critical because without treatment blindness will quickly occur. One study documented that stabilization of vision was limited to patients with initial visual acuity of 20/40 or better at the beginning of treatment (19). Although timely treatment of CMV retinitis is critically important, the logistics of CMV retinitis treatment, that is, complication ridden intravenous (IV) medications for the rest of the patient's life, represents not only a major inconvenience, but also an emotional mile stone in an eventually fatal disease. Death is sufficiently abstract so that many patients appear surprisingly cavalier about the prospects until they are very ill. Blindness, however, is more immediate and concrete with profound emotional impact. Therefore, clinicians must be very careful to correctly diagnosis CMV retinitis and yet be conservative about advising treatment for small patches of peripheral CMV retinitis (3). CMV retinitis can advance rapidly so these patients must be watched very carefully.

A regimen of intravenous (IV) virostatic ganciclovir and/or foscarnate is used to treat CMV retinitis. A loading dose is given and depending on clinical response a maintenance dose is instigated. Once IV ganciclovir and/or foscarnate is begun it must be continued indefinitely. Life expectancy after the discovery of CMV retinitis is approximately 12.6 months with foscarnet treatment and 8.5 months with ganciclovir treatment (26,27,28).

Intravitreal delivery of ganciclovir and foscarnate is possible, both by repeated intravitreal injections or by implanted intravitreal slow delivery devices (29,30,31). Both of these methods have the disadvantage of not offering treatment for systemic CMV disease or protection to the uninvolved eye. The third disadvantage is the discomfort, cost and risk of twice a week intraocular injections. Their advantage is to provide continuation of ocular treatment without systemic side effects. Although technology for intravitreal drug administration is improving their intrinsic disadvantages limits them except for special cases.

Oral ganciclovir capsules (CYTOVENE) are now available and are used for maintenance treatment after IV induction (32). Times to breakthrough (a term for recurring CMV retinits while on maintenance treatment) are somewhat shorter for oral maintenance regimens than for IV maintenance programs; however, the ease of administrating oral ganciclovir makes its use attractive. A long acting anti-CMV medication (HPMPC or Cidofovir) appears to be a promising drug for IV and intravitreal use (33). Resistance has not yet occurred during initial trials. Intraocular injection can be approximately 55 days apart. This represents a huge improvement over current intravitreal injections which must be repeated twice or more per week.

Retinal detachment is a potentially blinding complication of **healing** CMV retinitis in as many as 50% of treated individuals (34,35,36). Recognition of retinal detachment in a treatment responding eye is difficult and yet of great importance. Although the usual methods of treating retinal detachment such as laser retinopexy and/or scleral buckle are not as effective for CMV retinal detachments as they are for standard retinal detachments, stabilization of vision can be achieved in 80% or more by the use of intraocular silicone oil tamponade (36,37,38). Patients should be cautioned to report any loss of vision even after a good response to treatment.

Two conditions can be confused with CMV retinitis: Cytoid bodies with hemorrhage, and myelinated nerve fibers. Some cytoid bodies will have small spots of blood. However, cytoid bodies are single and smaller than CMV retinitis. In contrast to cytoid bodies pigment changes usually accompany CMV retinitis and the lesions are larger and occur along major vessels. The differential becomes difficult when CMV lesions are very early, single and accompanied by scant blood. As long as the suspect lesions do not threaten the fovea or optic nerve they can be watched. CMV retinitis will quickly enlarge with more hemorrhage where cytoid bodies will gradually fade.

Myelinated nerve fibers are congenital extensions of the normal nerve sheath (myelin) past the optic nerve into the retina. This normal variant will present as a white, feathery, superficial area of nerve fibers extending radially out from the optic nerve. There is never bleeding associated with myelinated nerve fibers and the lesions are stable (Photo #4).

2. Toxoplasmosis retinitis

Although CMV retinitis comprises the majority of opportunistic ocular infections many other disease entities can produce serious ocular disease. Toxoplasmosis is a common central nervous system

(CNS) opportunistic infection in immunocompromised patients and may be the initial manifestation of AIDS. Ocular toxoplasmosis is less common than CNS toxoplasmosis. Ocular toxoplasmosis is seen as a single or multifocal retinal lesion in one or both eyes or may be manifest as a massive retinal inflammation with retinal necrosis (39,40,41). Immunocompetent toxoplasmosis usually occurs as daughter lesions emanating from an original congenital pigmented chorioretinal "mother" scar. Toxoplasmosis in immunocompromised patients is a manifestation of acquired rather than congenital disease and instead of emanating from previous lesions the retinal inflammation and necrosis begins in a previously normal retina (39).

Anterior segment reaction in toxoplasmosis can be mild or quite severe depending on the severity of the disease. Clumps of vitreous cells can form "snowballs" often obscuring the posterior pole. Treatment is the same as for CNS toxoplasmosis and is effective in about 3/4 of the cases. Relapses occur without continued treatment. Treatment with systemic corticosteroids is **not** indicated and may cause exacerbation of CMV retinitis.

3. Herpes zoster

Acute retinal necrosis (ARN) is a devastating ocular complication of AIDS. This disease has been known to ophthalmology for some years and is seen occasionally in immunocompetent individuals. The organism is usually herpes zoster and occasionally herpes simplex. The AIDS type of ARN is particularly devastating and responds poorly to acyclovir and other antiherpetic agents (42,43,44) (Photo #5).

HIV-ARN occurs in patients with very low CD4 counts. It is manifest by loss of vision, eye pain and marked vitritis. There are well demarcated multifocal areas of necrotizing retinitis beginning in the periphery with occlusive vasculopathy. Other characteristics are optic atrophy and scleritis. Eighty percent of these individuals have had mucocutaneous herpes and many are bilateral (BARN). Most are associated with treatment failure and 69% result in blindness. With a combination of intravenous and intraocular antiviral medications long term remissions occur rarely (42,43,44).

In addition to the above, herpes zoster of the anterior sensory trigeminal ocular nerves (shingles) can produce chronic reccurring pan-uveitis and corneal disease. Vigorous treatment with acyclovir or other herpetic antivirals are proving to be effective in preserving vision in this situation.

"PORN" or progressive outer retinal necrosis syndrome is another rare necrotizing herpetic retinopathy, only recently recognized, that needs to be differentiated from CMV retinitis and acute retinal necrosis. This entity usually occurs after a bout of cutaneous herpes zoster (67%) and is likely to be a variant of herpes zoster infection (45). Visual loss is the first symptom with minimal anterior segment reactions. The retinal lesions are multiple, deep, and peripheral. The lesions coalesce to become confluent and even with aggressive antiviral treatment visual acuity is lost to "no light perception" (NLP) in most cases (46)(photo #5).

PORN emphasizes the point that the herpetic and other infectious entities have protean manifestations in immunocompromised individuals.

5. Pneumocystic carinii choroiditis

Pneumocystis carinii is a rare choroiditis that responds well to treatment. The lesions are multifocal, oval, smooth edged, occur deeper in the choroid and are easily differentiated from CMV retinitis (photo #2). This entity is almost always associated with the use of aerosolized pentamidine therapy for pneumocystic pneumonitis (47,48). Photo #2 is particularly valuable because it demonstrates pneumocystic choroiditis as well as CMV retinitis (a frosted angiitis presentation as well as a classical presentation) and non specific HIV retinitis.

Other organisms can cause choroiditis in immunocompromised individuals. Cryptococcus (48), Mycobacterium avium (5), Mycobacterium tuberculosis (4,49,50), secondary syphilis(51,52), Histoplasmosis (53), and others have been reported to involve the choroid.

6. Syphilis

Neurosyphilis and extraneural syphilis can effect all structures of the eye. In one study of nine patients syphilis was found to produce iridocyclitis, vitritis, retinitis and neuroretinitis, papillitis, optic perineuritis and retrobulbar optic neuritis (51,52). Syphilis has many manifestations both ocular and extraocular. In AIDS a high index of suspicion for ocular syphilis must be maintained. This is especially important since it is one of the more treatable opportunistic infections.

7. Non-Hodgkins lymphoma (NHL)

Systemic and CNS non-Hodgkins lymphoma (NHL) is 60 to 100 times more likely to occur in the AIDS population; however, primary ocular or orbital involvement is rare. Most ocular findings of NHL are those of secondary neurological disease from CNS lymphoma (26% of cranial neuropathies in one study{54}). Extraocular orbital lymphomas present as gradually progressive, unilateral (occasionally bilateral) proptosis with typical findings of a medial orbital wall mass with bony erosion on computerized tonography (55,56).

Intraocular NHL (reticulum or large cell lymphoma) is part of primary CNS lymphoma and can involve brain, meninges, spinal cord, and rarely the eye.

Intraocular large cell lymphoma can mimic many other intraocular diseases and is sometimes known as the "Masquerade Syndrome". The classical presentation of intraocular NHL is that of multifocal solid tumor-like retinal pigment epithelial infiltrates (57,58). Intraocular NHL can expand to include the retinal vasculature, vitreous and anterior segment resulting in a misdiagnosis of CMV, toxoplasmosis, herpetic or other intraocular microbial infection (58,59).

Diagnosis of intraocular NHL often depends on vitreous biopsy. Even this modality of diagnosis is difficult, demanding a special processing of samples. In that NHL responds well to treatment modalities of steroids and radiation, correct diagnosis is imperative.

B. Optic nerve

Direct optic nerve disease occurs commonly with AIDS secondary to a number of opportunistic infections (60). Cryptococcal meningitis is often associated with optic nerve disease; most often by papillitis (swelling of the ocular portion of the optic nerve) and optic atrophy (61). On rare occasions cryptococcal choroiditis with yellow choroidal infiltrates occurs in association with optic atrophy (61). Toxoplasmosis gondii, as well as acute retinal necrosis, can directly affect the optic nerve (62). CMV retinitis adjacent to the optic nerve can cause papillitis and eventually optic atrophy (22). Viral-associated intraocular tumors such as large cell lymphomas can directly involve the optic nerve. Syphilis and neurosyphilis can effect the optic nerve as well as other ocular structures (51,52).

C. Anterior segment

1. Infectious keratoconjunctivitis

A host of opportunistic ocular infections involve the anterior segment of the eye as well as retina and posterior structures. One study of 244 AIDS patients found: dry eyes (12%), blepharitis (9%), meibomitis (4%), bacterial conjunctivitis (3%), palpebral molluscum contagiosum (2%), herpes zoster ophthalmicus (1%), bacterial corneal ulcer (1%), mycotic corneal ulcer (1%), and mycotic conjunctivitis with plaque formation (1%). Kaposi's sarcoma was found in 4 patients and limbic cryptococcosis was found in one patient (63). Gonococcal infection must be considered in cases of supperative keratoconjunctivitis (64).

Microsporidial keratoconjunctivitis is being recognized as a cause of keratoconjunctivitis in immunodeficient individuals. Corneal epithelial irregularity with translucent, white punctate opacities in the corneal epithelium that responds to treatment with propamidine isethionate 0.1% (Brolene) has been reported(65,66). Microsporidial keratoconjunctivitis can be associated with extraocular disease such as sinusitis, enteritis, hepatitis and peritonitis (65,66).

Molluscum contagiosum, unusually seen in children, can infect the lids of adults in immunodeficient individuals with resultant conjunctivitis and keratitis (67).

Persistent blepharitis and keratoconjunctivitis that is unresponsive to aggressive dry eye therapy should be appropriately diagnosed and aggressively treated depending on culture and biopsy findings.

2. Corneal ulceration

Corneal ulceration in AIDS patients have been associated with a number of organisms. The list includes, but is not limited to: capnocytophagia, Candida albicans, alpha-hemolytic streptococci, Staphylococcus aureus, microsporidia, and gonococcus (68). Corneal ulcerations are uniformly very serious in immunocompromised patients and represent a medical emergency. The risk for corneal ulceration is higher with continuous wear contact lenses.

3. Kaposi's sarcoma and other skin and conjunctiva tumors

Kaposi's sarcoma can involve the lids and conjunctiva with elevated purple lesions (69). These are managed in a similar manner to Kaposi skin lesions elsewhere. Conjunctival squamous cell carcinoma occurs on rare occasions in AIDS patients and is particularly aggressive (70). Although these tumors are rare it is important to not ignore persistent or unusual lid or conjunctival growths in AIDS patients. Most of these tumors respond to appropriate treatment if diagnosed and treated early.

III. INDIRECT OCULAR DISEASE

Indirect ocular disease secondary to AIDS is usually neuro-ophthalmological and results from CNS disease. Any combination of ocular neurological signs and symptoms can occur for any single or combination of central nervous system infections, tumors or autoimmune conditions. The manifestations are numerous and protean (13). However; occasionally, neuro-ophthalmological manifestations associated with AIDS can be localizing and diagnostic. Mansour found in a review of 177 AIDS patients: five cases of ocular motor nerve palsies, two cases of papilledema, two cases of CMV optic neuritis, and one patient each with cortical blindness, conjugate gaze palsy, and altitudinal visual field defect. Among the CNS diseases thought to be associated with these neuro-ophthalmological defects were toxoplasmosis, lymphoma, cryptococcal meningitis, systemic CMV infection and herpes simplex encephalitis (71).

Single cranial nerve palsies require extra attention. They can occur early secondary to HIV itself. In fact, single cranial neuropathies should prompt HIV testing in at risk populations (71).

Cryptococcus is especially important in patients who

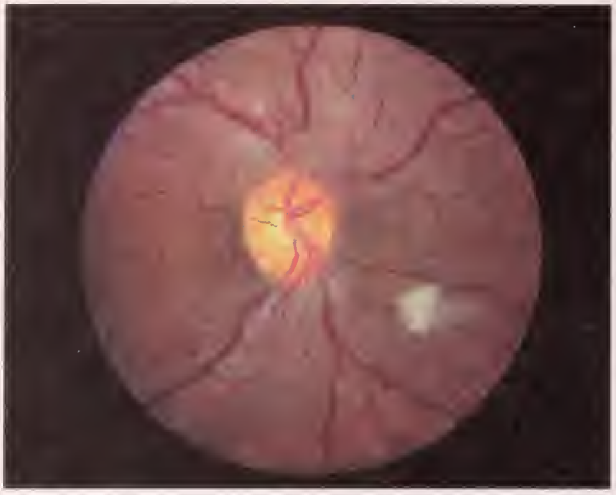

Photo #1. The fundus photograph shows the classical findings of non-specific HIV retinitis. Cytoid bodies or cotton wool spots are the white, opaque superficial lesions about one third of the diameter of the optic nerve head. Microaneurysms are the very small red dots throughout the posterior pole. The vascular congestion is more subtle than this reproduction can demonstrate. These three findings make up the ocular microangiopathic syndrome (OMS).

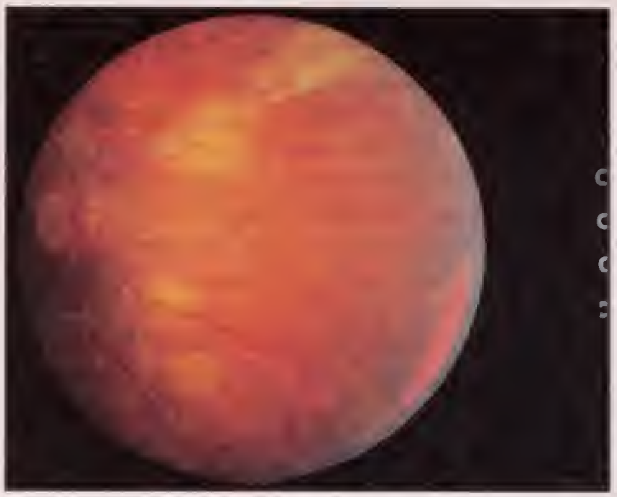

PHOTO #3. Demonstrates several aspects of opportunistic infections in immunocompromised individuals. The yellow, circular, smooth-edged, deep choroidal lesions are due to Pneumocystis cariini choroiditis. Anterior to this are the thick, white, hemorrhage lesions of CMV. Note the white, thick CMV around the vessel superiorly. This severe vasculitis is known as “frosted angiitis”.

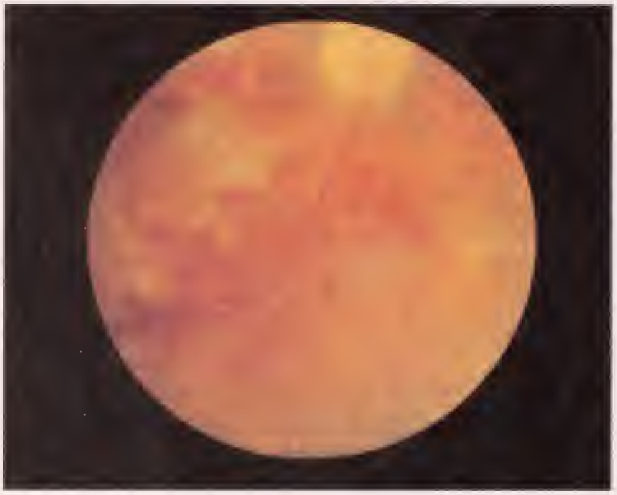

PHOTO #2. Demonstrates the white, thick, dry appearing, hemorragic, irregular inner retinal lesions of CMV retinitis.

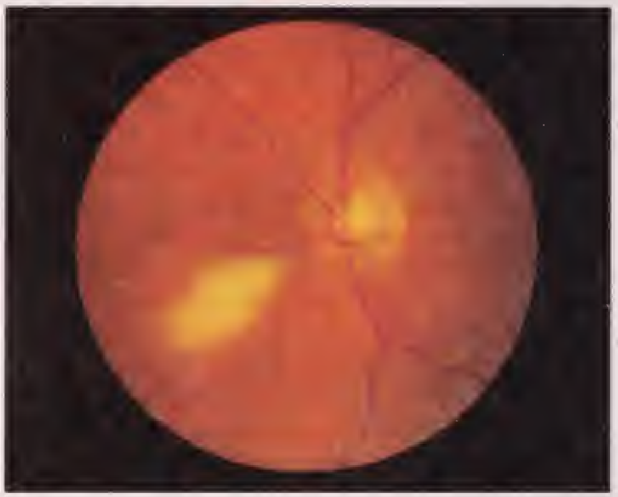

PHOTO #4. Shows a typical myelinated nerve fiber that can be confused with CMV retinitis. A cytoid body of HIV-OMS is seen just above the inferior arcade vessel on the right edge of the photograph.

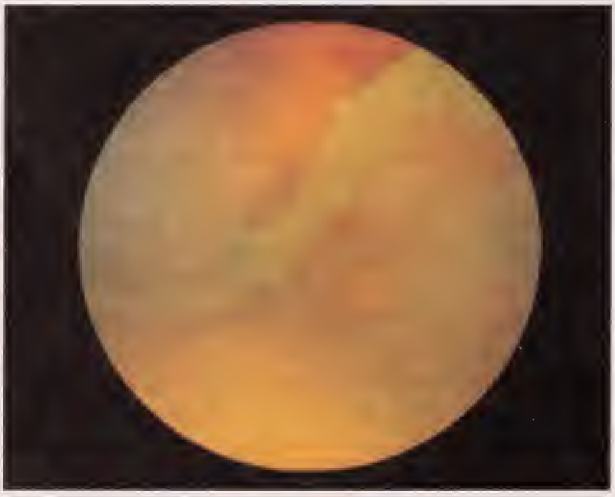

PHOTO #5. Demonstrates the severe retinal reaction to herpes zoster in the PORN syndrome. A large retinal tear can be seen running obliquely through the upper left quadrant. The retinal edge of the retinal tear is seen to be folded over toward the center.

have unexplained loss of vision with no ocular objective findings. Cryptococcus is common in AIDS occurring in 1.9% to 11% (1). Sudden simultaneously bilateral visual loss has been described. The exact mechanism of this catastrophic blindness is unknown, but may be due to focal necrosis of the optic nerves (72). Other explanations for bilateral vision loss due to cryptococcus are infiltration of the optic chiasm and optic tract, as well as adhesive arachnoiditis, intracranial hypertension and cerebral vasculitis. Other ocular symptoms that occur with cryptococcus meningitis are photophobia, blurred vision, and cranial nerve palsies.

IV. TOXIC DISEASE DUE TO AIDS MEDICATIONS

An additional ocular manifestation of AIDS that is often overlooked is ocular toxicity due to AIDS medications. For example, **ethambutol** (an anti-tuberculous drug) is neurotoxic and can result in non-reversible optic atrophy (73). **Amphotericin B** for fungal treatment has been reported to result in neural complications including optic atrophy. Systemic and topical corticosteroids can result in glaucoma as well as cataracts. **Anti-depressant** sympathomimetics exacerbate AIDS related keratoconjunctivitis sicca. Recent studies have shown an association between anterior uveitis and the combined use of **clarithromycin** and **rifampin** (74). The cause of this is thought to be clarithromycin enhanced absorption of rifampin which in turn produces the uveitis. Topical treatment is usually effective and rifampin need not be discontinued. Lower doses of rifampin to reflect additional absorption is usually sufficient.

Corneal toxicity to drugs that affect lysosomes such as **ganciclovir** or **AZT** may cause corneal epithelial lipidosis (a central corneal punctate epitheliopathy) with a similar appearance to that seen with chloroquine or amiodarone ocular toxicity. This toxicity can be mistaken for microsporidial keratoconjunctivitis (75). As new drugs are made available and as we gain greater experience with current medications we need to remain vigilant for ocular toxicity.

V. REFERRAL GUIDE (OPHTHALMOLOGIST)

The question of appropriate ocular specialist referral is very important. Thoughtful consideration of general principals of AIDS ocular complications may be helpful when applied to the question of ophthalmology referral. **First**, ocular complications of AIDS in patients with CD4 counts of >100 consist of nonspecific retinopathy (OMS), keratitis sicca, early onset presbyopia and mild optic atrophy. Except for reading glasses for presbyopia and dry eye care there is no treatment for these complications. **Second**, although symptoms usually occur with opportunistic ocular infection in patients with CD4 counts below <100 they may be very subtle, mimic normal aging processes and may be overlooked by patients with reduced cognitive abilities and denial. **Third**, opportunistic and indirect AIDS ocular complications in patients with CD4 counts <100 and especially <50 become very difficult to diagnose. Further, treatment decisions at this level become complex and are rapidly changing.

Considering the above facts current thinking (76,77), suggests an ocular specialist (ophthalmologist) referral for all HIV infected patients with:

A. CD4 counts <100 (with or without symptoms);
B. Symptoms of persistent visual change or loss;
C. Systemic or intracranial diseases known to be associated with ocular disease.

VI. CONCLUSIONS

The preceding has been an overview of our current understanding of ocular disease associated with HIV infection and AIDS. It is meant to be used as a reminder of the more common ocular diseases encountered with an emphasis on treatable and diagnostic disease. It is hoped that it will also be a reminder that ocular disease is a complex, common, and particularly devastating aspect of AIDS. Early recognition of potentially blinding treatable disease will be of great benefit to these unfortunate individuals.

Following is an outline of ocular disease associated with HIV infection and AIDS.

HIV AND AIDS OCULAR DISEASE OUTLINE

I. Nonspecific (seen earlier with relatively higher CD4 counts}
  A. OMS (ocular microangiopathic syndrome)
    1. Cytoid bodies
    2. Microaneuryms
    3. Intraretinal hemorrhage
  B. Optic nerve atrophy
  C. Vitreous cells
  D. Uveitis
  E. Keratitis sicca (dry eyes)
  F. Premature presbyopia
II. Opportunistic infections
  A. Posterior segment
    1. CMV retinitis
    2. Toxoplasmosis retinitis and vitritis
    3. Herpes zoster (ARN ,BARN and PORN)
    4. Pneumocystis choroidopathy
    5. Syphilis
    6. Viral associated choroidal tumors such as non-Hodgkin's (large cell or reticulum cell) lymphoma

B. Optic nerve disease
   1. Nonspecific HIV optic atrophy
   2. Cryptococcus
   3. Toxoplasma
   4. CMV
   5. Non-Hodgkin's lymphoma (NHL)
   6. Syphilis

C. Anterior segment
   1. Infectious keratoconjunctivitis (from multiple organisms)
   2. Corneal ulcerations
   3. Kaposi's sarcoma and other skin and conjunctival tumors.
   4. Carcinoma
   5. Non-Hodgkin's lymphoma (NHL)

III. Indirect Ocular Disease
   A. Mostly due to intracranial neurological diseases.
   B. Single cranial nerve palsies
   C. Cryptococcus blindness

IV. Toxic reactions to AIDS medications
   A. Ethambutol
   B. Corticosteroids
   C. Antidepressants
   D. Reframpin/clarithromycin uveitis
   E. Lysosome affecting drugs

V. Referral guide (ophthalmologist)
   A. CD4 counts <100 (with or without symptoms).
   B. Symptoms of persistent visual change or loss.
   C. Systemic or intracranial disease known to be associated with ocular disease.
   D. Suspected intracranial disease.

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Health Effects of Methyl Tertiary Butyl Ether (MTBE) in Gasoline in Alaska

Mary Ellen Gordian, M.D., M.P.H.(1)
Michael D. Huelsman (1)
Mary-Lynn Brecht, Ph.D. (2)
Dennis G. Fisher, Ph.D. (3)

ABSTRACT

Objectives. This ecologic study assessed whether there was a change in health status in Alaska in the winter of 1992-93 after the introduction of MTBE in gasoline. Methyl tertiary butyl ether(MTBE) is used as a fuel oxygenate in the United States and in Europe. In the winter of 1992-93 MTBE was added to gasoline in the cities of Fairbanks and Anchorage, Alaska. The program was discontinued in Fairbanks in December, 1992, but continued in Anchorage until February 28,1993.

Methods. Outpatient visits for state employees and dependents (n=~28,000) living in Alaska were compared over three winters by analyzing health insurance claims.

Results. Odds ratios were calculated. The odds ratios indicated that the winter of 92-93 was not statistically different from previous winters in numbers of claims for upper respiratory illness, bronchitis, headache, or asthma in either Anchorage or Fairbanks.

Conclusion. There was no increase in claims for respiratory illness in either city after introduction of MTBE.

INTRODUCTION

In the fall of 1992 several cities in the United States including Anchorage and Fairbanks, Alaska, were mandated to use oxygenated fuel under the 1990 Clean Air Act Amendments. On October 15 Fairbanks complied with this requirement by adding 16% methyl tertiary butyl ether (MTBE) to gasoline sold in that city. Anchorage started the program on November 1, 1992.

MTBE has been used as a gasoline additive in increasing concentrations in the United States and Europe to reduce carbon monoxide emissions and ozone formation (1,2). There were no previously noted health problems with its use although extensive study of health effects in humans had not been done.

Fairbanks residents voiced concern about the addition of MTBE to gasoline on Talk Radio and a hot-line was set up to document complaints. Complaints included increased cost ($.14/gallon), increased odor, mechanical problems, reduced gas mileage, and increased headaches and respiratory problems associated with gasoline exposure.

In mid-December 1992, MTBE was taken out of the gasoline sold in Fairbanks after public complaints of adverse health effects and concerns raised by a study done by Center for Disease Control and Prevention (CDC) and the Alaska State Department of Health and Social Services (DHSS) (3). A telephone survey done as part of that study in December found that 71% opposed the use of oxy-fuel, 65% attributed health complaints to the use of oxy-fuel, and 25% of those had sought medical care for their symptoms. (Moolenaar and Heflin, letter to Department of Health and Social Services, Division of Epidemiology, Dec 10,1992.)

The controversy received national media coverage and EPA convened a conference in Falls Church, Virginia in July 1993, to discuss studies done to determine whether MTBE was affecting people's health. As MTBE is being used worldwide in reformulated gasoline, the issue of whether MTBE increases respiratory problems was considered high priority.

Chamber studies of exposures to MTBE did not elicit the symptoms that were reported in Fairbanks (4). CDC

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(1) Department of Health and Human Services, Municipality of Anchorage, Anchorage, Alaska.
(2) School of Nursing, University of California, Los Angeles, California.
(3) Alcohol and Addiction Studies, University of Alaska Anchorage.

Correspondence and reprints
Mary Ellen Gordian, MD
Department of Health and Human Services
Municipality of Anchorage
P.O. Box 196650
Anchorage, AK 99519-6650

and Environmental and Occupational Health Sciences Institute (EOHSI) studies done in New York, Connecticut, and New Jersey showed the same level of symptoms among service station attendants regardless of whether MTBE was used as an oxygenate or not (5,6,7). However, the power of these studies may not have been sufficient to detect a small effect. There were also many questions raised about the impact of the climate of Alaska on human response to MTBE.

The composition of Alaska gasoline was also found to be significantly different from gasoline used in other areas of the country. Duffy at University of Alaska Fairbanks determined that the odor threshold of Alaskan gasoline was significantly reduced when MTBE was added to it (8). People were more likely to be able to smell Alaska gasoline after MTBE was added.

The purpose of this study was to determine whether there was an increase in medical visits in Anchorage and Fairbanks in the winter of 1992-93. This retrospective study used health insurance claims for outpatient care as a surrogate measure of illness in Alaska. Use of claims data for population based health research is increasing because of the large data base availability and low cost of claims information (9). Although diagnoses from insurance claims data are not always reliable for research on outcomes or procedures because of missed or inaccurate diagnoses, the simpler the procedure the more reliable the data (10). Outpatient claims are usually coded directly by the provider of care at the time of the visit and therefore should be the most reliable.

METHODS

Computerized data were available from Aetna insurance company for State of Alaska employees and retirees and their dependents for whom numbers of insured persons by zip code of residence were available. State of Alaska insurance benefits ($100 deductible per individual, $300 per family per year, 90% coverage for outpatient care) for employees and dependents had remained constant since 1989. Supplemental insurance further reducing deductibles was elected by the same percentage of employees each year.

The insurance company provided reports of claims for outpatient care including office visits, emergency room visits, and home visits; but excluding laboratory and x-ray department visits, and inpatient admissions. The ICD-9 codes were obtained by asking practicing physicians which ICD-9 codes they ordinarily use for the kinds of symptoms alleged to be associated with MTBE. ICD-9 codes are fairly well standardized in the community with most physicians using standard superbills or computer programs.

Individual identifiers were not used so numbers of visits for the diagnoses, not individuals, are counted as cases. Only Alaska zip codes for both provider and patient were used. The specific months that MTBE was sold in Anchorage were: November, December, January, and February. We were limited by the availability of computerized reports to the last three years.

The initial investigation in Fairbanks described a case as someone having one or more of the following symptoms: headache, nausea, cough, throat and eye irritation (1). The diagnoses were grouped into upper respiratory conditions, lower respiratory conditions (excluding pneumonias), asthma and asthma-like symptoms, and headaches. All ICD-9 codes for each condition are grouped together. Nausea is a common symptom for a wide variety of conditions suggesting many diagnoses. An increase in nausea can not be evaluated by this method.

The insurance reports were divided by zip code into three areas of the state, Anchorage , Fairbanks, and all others. Anchorage and Fairbanks sold gasoline enriched with 16% MTBE in the winter of 1992-93. Fairbanks ceased requiring MTBE on December 15, 1992. However, because any MTBE gasoline still present in inventory was sold and used, Fairbanks could still be considered an MTBE area for the month of December, 1992.

The number of claims for people treated for respiratory symptoms, asthma, and headache during the winter of 1992-93 in the Anchorage area is compared to the number of claims for treatment for similar symptoms in the two proceeding winters.

ANALYSIS

The rate of illness was calculated on the basis of claims made each month divided by the number of members with Aetna State Employees coverage in this area. A mean value for each winter for each area was calculated. The number of active employees and dependents in Anchorage was about 10,000, Fairbanks was 3000, and other Alaskan members outside of Anchorage and Fairbanks was about 13,000. The number of retirees and dependents was 5000 in Anchorage, 1900 in Fairbanks, and 7000 in other parts of Alaska. Retirees living out of state were not included.

The odds ratios with 95% confidence intervals were calculated comparing the winter of 1992-93 versus 1991-92 and 1990-91 in Anchorage and Fairbanks (Table 1). The odds ratio for Anchorage used number of claims per population covered over a four month period November through February. The calculation for Fairbanks compared only the months that MTBE was actually in use in Fairbanks, November and December, to previous claims experience for the same two months. The claims for retirees were analyzed separately, and showed similar results.

Table 1

Active Employees and Dependents				
Anchorage*			Fairbanks**	
Odds Ratio	C.I.95%	Upper Respiratory Illness	Odds Ratio	C.I. 95%
0.94	0.84-1.05	92-93 vs. 90-91	0.95	0.81-1.13
0.94	0.84-1.05	92-93 vs. 91-92	1.07	0.90-1.27
1.00	0.90-1.12	91-92 vs. 90-91	0.89	0.75-1.06
		Bronchitis		
0.90	0.73-1.11	92-93 vs. 90-91	1.33	0.90-1.99
0.85	.069-1.05	92-93 vs. 91-92	0.96	0.67-1.38
1.06	0.86-1.30	91-92 vs. 91-91	1.39	0.93-2.07
		Asthma		
1.05	0.76-1.47	92-93 vs. 90-91	1.16	0.75-1.81
0.99	0.72-1.37	92-93 vs. 91-92	1.00	0.66-1.52
1.06	0.76-1.49	91-92 vs. 90-91	1.16	0.75-1.82
		Headaches		
1.54	0.94-2.52	92-93 vs. 90-91	1.43	0.71-2.94
0.52	0.35-0.75	92-93 vs. 91-92	1.30	0.67-2.58
2.94	1.89-4.68	91-92 vs. 90-91	1.10	0.52-2.36

* Four months of winter - November-February
** November and December only

RESULTS

The results indicate that the rate of claims for illness in the winter in Anchorage and Fairbanks has been stable over the last three years. The rate of claims in the winter of 1992-93 in both Anchorage and Fairbanks peaked in December, the month that the surveys were done by the Alaska State Health Department. The rate of illness decreased in January and February in both Anchorage and Fairbanks even though MTBE was still being used in Anchorage. There was no increase in claims for illness in Anchorage or Fairbanks during the winter of 1992-93. The only category for retirees which had sufficient numbers to allow comparison was upper respiratory illness for which the confidence interval of the odds ratio included 1.0 for all comparisons between winters.

DISCUSSION

The peak in headaches in January 1992 was remembered by providers as an epidemic of a viral illness that caused headaches. This ability to detect epidemics albeit retrospectively gives credence to this method of evaluating the possibility of an increase in respiratory symptoms or headaches due to a new environmental agent.

This study assumes that the rates of illness are proportional to the rates at which people use medical services and that the use of medical services will be reflected in claims made for insurance. Although we are looking at visits and not individuals, by grouping ICD-9 codes into general areas, the numbers become large enough not to be unduly influenced by the care-seeking behavior of any one individual.

Mild effects of short duration that were only irritating but not associated with increased health problems could be missed by this study. Other factors which might reduce the apparent rate of illness was the possibility that a patient being treated for another medical problem might also complain to their physician about respiratory problems and that complaint would not be the diagnosis reported for the visit.

The power of this study is high enough to determine a one percent change in claims per month at 91% confidence for a two-tailed test of differences between proportions at an alpha of 0.05.

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(continued on page 119)

Composite Tabulation of Deaths in Anchorage, 1985 - 1988

Rodman Wilson, M.D.[1]

A series of reports in this journal tabulated numbers and causes of death in Anchorage, Alaska, for the years 1985, 1986, 1987, and 1988 (1-4). Deaths of residents of Anchorage dying away from home 1985-88 were also collected (5).

The mean estimated population of Anchorage in these years was 235,575 persons - 43.7% of the mean population of 539,425 of the state. The 3,435 deaths during the quadrennium comprised 41.1% of the 8,351 deaths in Alaska during the years 1985-88 (6).

Autopsy was done in 35.1% of cases.

Although the final report of deaths (4) gave selected data for deaths over the four-year period, consolidated listing of all deaths was not published.

Remember that I edited causes of death by scrutinizing all words on each certificate of death, talking to attending physicians and pathologists (20.8% of cases), and then assigning an underlying cause of death to the case. In many instances this differed from the underlying cause listed by the physician at the time of death. The latter, however, remained the official cause of death for state and national statistical purposes. Examples of altered causes of death were displayed in a previous report (4, Table 1).

I submit, as argued before (1-4), that the refined, corrected, and edited listings of underlying causes of death are closer to the true causes of death among these 3,435 cases than the initial recordings of the attending physicians.

Here is the composite table of all deaths in Anchorage, 1985-88. Incorporated are minor corrections in numbers previously given and a few further alterations in final cause of death in light of later information, such as homicide being proved.

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Composite Tabulation

Underlying Causes of Death of 3,435 Persons Dying in Anchorage, Alaska, 1985-1988

(Code numbers are from ICD-9)

CARDIOVASCULAR DISEASE				**995**	**(29.0%)**
Heart Disease*			686		
coronary atherosclerosis (410,414)		591			
acute myocardial infarction (410)	238				
cardiomyopathy, idiopathic (425.4)		23			
rheumatic (393-398)		19			
aortic valve stenosis, idiopathic (424.1)		14			
hypertensive (402, 404)		14			
congenital (745-746)		5			
conduction defect, various (426, 427)		3			
cardiac arrest, unexplained (427.4, 427.5)		2			

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[1] Dr. Wilson is an internist. He is currently acting executive director of ASMA.

primary pulmonary hypertension (416.0)		2			
hypertophic obstructive cardiomyopathy (425.1)		2			
mitral valve incompetence, unspecified (424.0)		2			
ruptured mitral chordae tendineae (429.5)		2			
type undertermined or unspecified (429.9)		7			

*excluding alcoholic cardiomyopathy, infections of the heart and death from heart disease before age 1

Vascular Disease *			309		
cerebral thrombosis (434.0)		142			
cerebral hemorrhage		60			
subarachnoid (430)	30				
intracerebral (431)	27				
arteriovenous malformation (747.81)	3				
aneurysm of aorta		30			
abdominal (441.3, 441.4)	26				
dissecting (441.0)	2				
thoracic (441.1)	2				
pulmonary embolism (415.1)		29			
atherosclerosis, generalized (440.9)		22			
mesenteric artery thrombosis (557.0)		12			
atherosclerosis, extremities, not diabetic (440.2)		5			
cerebral embolism (434.1)		3			
aortoiliac obstruction (444.0)		3			
atherosclerosis, carotid artery (433.1)		1			
aneurysm of iliac artery (442.2)		1			
aneurysm of femoral artery (442.3)		1			

*excluding cerebral hemorrhage before age 1 and diabetic gangrene of extremities

CANCER				**860**	**(25.0%)**
Lung (162)			275		
Breast (174)			77		
Colon and Rectum (153, 154)			74		
Pancreas (157)			39		
Metastatic, origin undetermined (199.0)			39		
Brain (191)			37		
Lymphoma (200-202)			34		
Hodgkin's Disease (201)		5			
Prostate (185)			31		
Ovary (183)			23		
Stomach (151)			22		
Melanoma (172)			21		
Bladder (188)			20		
Myeloid Leukemia (205)			19		
Oropharynx (146-149)			16		
Kidney (189)			16		
Esophagus (150)			15		
Cervix Uteri (180)			12		
Multiple Myeloma (203)			10		
Tongue (141)			10		
Lymphoid Leukemia (204)			7		
Gall Bladder (156.0)			6		

Bile Duct (235.3)		5		
Liver, primary (155.0)		5		
Larynx (161)		5		
Osteosarcoma (170)		5		
Sarcoma, soft tissue (171)		4		
Endometrium (182)		4		
Myeloproliferative Disorder (238.7)		3		
Embryonal Cell (186)		2		
Esophagogastric Junction (151.0)		2		
Duodenum (152.0)		2		
Anus ((154.3)		2		
Penis (187)		2		
Mastoid, squamous cell (160.1)		1		
Parotid (142.0)		1		
Gastrinoma (157.9)		1		
Carcinoid (235)		1		
Ampulla of Vater (156.2)		1		
Adrenal (194.0)		1		
Ureter (189.2)		1		
Choriocarcinoma (181)		1		
Vagina (184.0)		1		
Pericytoma (158.9)		1		
Polycythemia Rubra Vera (238.4)		1		
Hairy Cell Leukemia (202.4)		1		
Plasma Cell Disease, macroglobulinemia (203.8)		1		
Angioimmunoblastic Lymphadenopathy (202.9)		1		
Liposarcoma (171.3)		1		
Spinal Cord (192.2)		1		
VIOLENCE AND ADVERSE EFFECTS			**715**	**(20.8%)**
Alcoholism		173		
alcoholic liver disease (571.0-571.3)	100			
acute alcoholism (303.0)	26			
alcoholic cardiomyopathy (425.5)	13			
hypothermia, drunk ((E901.0)	11			
alcoholic pancreatitis (577.0, 577.1)	7			
chronic alcoholism (303.9)	7			
head injury or fall, drunk (E835, E880, E888)	5			
delirium tremens (291.0)	2			
abscess of lung, aspiration, drunk (513.0)	2			
Vehicular Impact		170		
automobile, truck (E811, E812, E815, E816)	97			
auto-pedestrian (E814)	38			
aircraft (E841)	12			
motorcycle and motorcycle-auto (812.2, 812.3)	5			
fall under moving truck (E818)	3			
auto-bicycle (E813)	3			
bicycle (E826)	2			
train-pedestrian (E805.2)	2			
boating or rafting, impact (E831)	2			
all-terrain vehicle (E821)	2			
3-wheeler-aircraft (E844)	1			
snowmachine (E820)	1			
sled-auto (E813)	1			
sled-tree (E917)	1			

Suicide		**145**
gunshot (E955)	84	
hanging (E953.0)	20	
motor vehicle exhaust ((E952.0)	19	
drug or chemical (E950)	16	
cutting wrist (E956)	2	
jumping (E957)	2	
drowning (E954)	1	
fire (E958.1)	1	
Homicide		73
gunshot (E965)	50	
cutting, stabbing, hatcheting (E966)	9	
strangling or suffocation (E963)	6	
bludgeoning (E967, E968.2)	5	
fist (E960)	1	
cocaine injection (E962.0)	1	
unknown means (E968.9)	1	
Poisoning, unintentional		32
cocaine (E855.2)	19	
methanol (E860.2)	4	
heroin (E850.2)	2	
morphine (E850.2)	1	
lidocaine (E855.2)	1	
tricyclic antidepressant (E850.4)	1	
multiple drugs (E858.8)	1	
fluorinated hydrocarbon aerosol (E869.8)	1	
insect repellent (E980 .7)	1	
unknown drug (E858.9)	1	
Fire (E980)		27
Drowning (not incl suicide by drowning) (E910)		19
Injury on Job		18
fall (E880-E888)	7	
crush (E916, E919)	5	
struck by object ((E917, E919)	5	
electrocution (E925)	1	
Fall, not on job, not drunk (E880-E888)		15
Gunshot, unintentional (E922)		12
Undetermined whether suicide, homicide or unintentional		10
gunshot (E985)	2	
motor vehicle exhaust (E982.0)	2	
prescription drugs (E980)	2	
cocaine (E980.4)	1	
hanging (E983.0)	1	
blow to chest (E988.8)	1	
injury, undetermined (skeletal remains) (E988.9)	1	
Mechanical suffocation, unintentional (E913)		6
Miscellaneous		15
crush, cave-in (E913,E916)	5	
blow, unintentional (E917)	3	
hit by ice falling from roof (E916)	1	
head injury, unexplained (E917)	1	
tear in trachea, unexplained (E920.9)	1	
electrocution, hang-gliding (E925)	1	
explosion, motor home (E825)	1	
avalanche (E909)	1	
hypothermia, disorientation after taking prescription drugs (E901.0)	1	

DEATH BEFORE AGE 1*		**254**	**(7.4%)**
Immaturity (765)	96		
Sudden Infant Death Syndrome (798.0)	52		
Congenital Anomalies (740-759)	43		
Birth Asphyxia (768)	8		
Respiratory Distress Syndrome (769)	8		
Hydrops Fetalis (778.0)	5		
Bacterial Meningitis (320)	5		
Pneumonia (480-486)	5		
Cerebral Hemorrhage, Neonatal (767.0)	3		
Aspiration of Meconium (770.1)	3		
Bronchopulmonary Dysplasia (770.7)	2		
Other Respiratory Conditions of Newborn (770)	2		
Fetal Alcohol Syndrome (760.71)	2		
Septicemia (036.3,038)	2		
Viral Myocarditis (422)	2		
Necrotizing Enterocolitis (777.5)	2		
Infantile Spinal Muscular Atrophy (335.0)	2		
Prolapse of Umbilical Cord (762.4)	1		
Endocardial Fibroelastosis (425.3)	1		
Congenital Heart Failure (779.8)	1		
Bacterial Endocarditis (421.0)	1		
Reye's Syndrome (331.81)	1		
Volvulus of Small Intestine (560.2)	1		
Congenital Hypoplastic Anemia (284.0)	1		
Oxalosis (271.8)	1		
Connective Tissue Tumor, perineum, benign (215.6)	1		
Psychomotor Retardation (318)	1		
No Neurologic Activity (779.0)	1		
Unknown Cause (799.9)	1		

*excluding 1 death from brain tumor and 9 from violence (unintentional asphyxia 3, fire 2, drowning 2, homicide 2)

LUNG DISEASES*		**214**	**(6.2%)**
Chronic Obstructive Pulmonary Disease⁺ (496)	165		
Asthma (493)	14		
Idiopathic Fibrosing Alveolitis (516.3)	8		
Tuberculous Fibrosis (011.4)	7		
Bronchiectasis (494)	4		
Bronchopulmonary Dysplasia (770.7)	4		
Rheumatoid (714.81)	2		
Restrictive, secondary to kyphoscoliosis (737.3)	2		
Tuberculosis (011)	1		
Sarcoidosis (135)	1		
Asbestosis (501)	1		
Silicosis (502)	1		
Byssinosis (504)	1		
Eosinophilic (518.3)	1		
Churg-Strauss Disorder (799.8)	1		
Hypoventilation, secondary to obesity (278)	1		

*excluding pneumonia, lung abscess, pulmonary embolism, and pulmonary disorders causing death before age 1

⁺COPD was listed as causing or contributing to death 302 times (8.8%) among 3,435 deaths.

INFECTION*			**147**	**(4.3%)**
Pneumonia (480-486)		51		
Acquired Immunodeficiency Syndrome (042)		24		
Septicemia (038)		18		
Viral Hepatitis		14		
non-A, non-B (070.4,070.5)	8			
B (070.2,070.3)	5			
A (070.0)	1			
Viral Myocarditis (422)		7		
Bacterial Endocarditis (421)		7		
Bacterial Meningitis		6		
pneumococcal (320.1)	3			
hemophilus (320.0)	2			
serratia (320.8)	1			
Pyelonephritis (590.0,590.80)		6		
Viral Encephalitis (049.9)		4		
Diverticulitis of Colon (562.11)		3		
Chickenpox (052)		1		
Acute Epiglottitis (464.3)		1		
Miliary Tuberculosis (018)		1		
Abscess, lung (513)		1		
Abscess, liver, Echinococcus multilocularis (122.5)		1		
Abscess, abdominal, cause undetermined (569.5)		1		
Botulism (005.1)		1		

*excluding death from infection before age 1, pulmonary tuberculosis, and abscess of lung secondary to aspiration, drunk

NEUROLOGIC DISEASE*			**106**	**(3.1%)**
Senile Dementia and Alzheimer's Disease (290.0, 331.0)		37		
Parkinson's Disease (332)		13		
Multi-Infarct Arteriosclerotic Dementia (290.4)		12		
Epilepsy (345)		10		
Amyotrophic Lateral Sclerosis (335.20)		6		
Encephalopathy, chronic, cause uncertain (348.3,507)		4		
Multiple Sclerosis (340)		3		
Guillain-Barré Syndrome (357.0)		3		
Hydrocephalus, congenital (742.3)		2		
Porencephaly, congenital (742.4)		2		
Metachromatic Leukodystrophy (330.0)		2		
Infantile Cerebral Palsy (343,343.2)		2		
Progressive Bulbar Palsy (335.22)		1		
Pseudobulbar Palsy (335.23)		1		
Psychomotor Retardation (318.2)		1		
Huntington's Chorea (333.4)		1		
Duchenne Progressive Muscular Atrophy (359.1)		1		
Syringomyelia (336.0)		1		
Demyelinating disease of CNS, unspecified (341.9)		1		
Pena-Shokeir Syndrome (759.8)		1		
Sleep Apnea (780.53)		1		
Psychosis, organic, not classified (294)		1		

*excluding cerebral thrombosis, cerebral hemorrhage, brain and spinal cord tumors, and death from neurologic disease before age 1

GASTROINTESTINAL DISORDERS*			**54**	**(1.6%)**
Acute Pancreatitis, non-alcoholic, (577.0)		10		
Gastric Ulcer		6		
bleeding (531.0)	3			
perforated (531.1)	2			
with adult respiratory distress syndrome (531.9)	1			
Acute Cholangitis (576.1)		4		
Acute Appendicitis (540)		4		
Intestinal Obstruction due to adhesions (560.81)		4		
Incarcerated Hernia (550.1,552.1,552.2)		4		
Volvulus		4		
small intestine (560.2)	2			
stomach (537.8)	1			
colon (560.2)	1			
Esophagitis (530.1)		3		
Duodenal Ulcer		3		
perforated (532.1)	2			
bleeding (532.0)	1			
Gastrointestinal Bleeding, cause undet (578.9)		2		
Diverticulitis of Colon, perforated (562.11)		2		
Perforation, intestine, cause undet (569.83)		2		
Hemorrhagic Gastritis (535.5)		1		
Ulcerative Colitis, acute (556)		1		
Cirrhosis of Liver, non-alcoholic (571.5)		1		
Cholelithiasis (574)		1		
Cholecystitis, acute (575.0)		1		
Inanition, secondary to short bowel syndrome (579.3)		1		

*excluding death from G-I disease before age 1

MISCELLANEOUS DISORDERS			**90**	**(2.6%)**
Diabetes Mellitus*		28		
end-stage renal disease (250.4)	18			
hypoglycemic coma (251.0)	3			
gangrene of extremities (250.7)	3			
ketoacidosis (250.1)	1			
hyperosmolar non-ketotic coma (250.2)	1			
complication unspecified (250)	2			
End-Stage Renal Disease, not diabetic (582-586)		11		
Senility (797)		8		
Aspiration and Aspiration Pneumonia, not drunk (E911, 507.0)		6		
Rheumatoid Arthritis (714, 720.0)		4		
Medication Toxicity (E870.0, E931.8, E944.7 E936)		4		
Systemic Lupus Erythematosus (710.0)		3		
Benign Prostatic Hypertrophy, obstruction (600)		2		
Panhypopituitarism (253.2)		2		
Hyperparathyroidism (252.0)		1		
Cystic Fibrosis (277.0)		1		
Immunodeficiency, congenital (279.0)		1		
Niemann-Pick Disease (272.7)		1		
Alpha 1-Antitrypsin Deficiency (277.6)		1		
Sickle Cell Anemia (282.6)		1		
Aplastic Anemia (284)		1		
Sideroblastic Anemia (285.0)		1		
Myelofibrosis (289.8)		1		

Raynaud's Disease (443.0)	1	
Relapsing Polychondritis (710.8)	1	
Systemic Sclerosis (710.1)	1	
Tuberous Sclerosis (759.5)	1	
Hemolytic Uremic Syndrome (283.1)	1	
Decubitus Ulcers (707.0)	1	
Obesity, gastroplasty (278.0)	1	
Inanition (263.8)	1	
Undetermined Cause (798,799)	5	

*Diabetes mellitus was listed as causing or contributing to death 186 times (5.4%) among 3,435 deaths.

	3,435	(100.0%)

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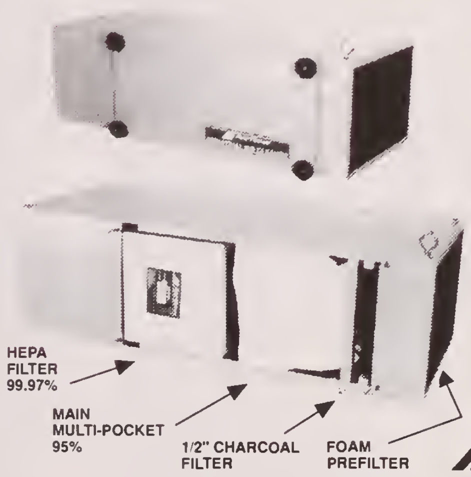

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Case History

BREAST ASYMMETRY: UNUSUAL PRESENTATION

Manuel A. Rodriguez, M.D.(1)

The patient is a 30-year-old, white female flight mechanic, who is gravida 0, not sexually active, and virginal. She had menarche at age 16 and has always had irregular periods, from every few months to having long periods of amenorrhea up to a year. She complained of irregular bleeding and breast asymmetry.

She has an interesting past medical history in that in 1975 she was involved in a motor vehicle accident. She was 12 years old at the time and had a significant head injury resulting in a coma and amnesia. She has no memory of the time prior to the accident. In addition to her amnesia, she had some motor abnormalities, but they gradually improved and over time was functionally normal without seizures or other apparent abnormalities. She has an identical twin who is healthy. Because of her past history and her history of oligomenorrhea, she has undergone gynecological evaluations in the past. She was treated with Premarin and Provera once to cause cyclic bleeding and she states that following this treatment she underwent dramatic bilateral breast enlargement. When she stopped the medication on her own, she states her left breast regressed, but her right breast remained large. Her breast asymmetry is of concern to her, because she is convinced that people are looking at her and interpreting this as abnormal. She feels well otherwise and is not concerned about her amenorrhea.

Past evaluations include a number of hormone assays. She had a TSH of 1.2, T4 of 7.4, and PRL of 24.9 which was slightly elevated, but was repeated with 18.4 normal result. She also had normal gonadotropins. Most recently she has been having irregular bleeding episodes every few months of moderate amount of flow.

Examination revealed a tall thin white female with normal blood pressure, pulse, and weight. HEENT, thyroid, heart, lungs, and abdomen were normal. Skin showed no hirsutism. Breasts showed moderate asymmetry, right greater than left. There were no masses or galactorrhea. Pelvic examination revealed normal external genitalia, vagina, and cervix. Introitus and vagina apparently virginal. Cervix was nulliparous. Bimanual exam was performed under anesthesia and revealed no adnexal masses. The uterus was normal size, anterior, and mobile. Vagina appeared well estrogenized and cervical mucous was present. Endometrial biopsy showed proliferative phase endometrium, sounded to approximately 6 cm. Other lab studies revealed LH, FSH, TSH, PRL, DHA-S, 17OH-P, AM Cortisol, testosterone, free testosterone, chem screen, and CBC within normal limits. P4: 0.50, was not indicative of ovulation. E2:43: was low.

DISCUSSION

In summary, this patient has oligomenorrhea, most likely due to hypothalamic etiology. One would be inclined to use the diagnosis of hypothalmic amenorrhea, but she must intermittently activate some since she has spontaneous periods from time to time indicating ovarian activation and estrogen production and endometrial proliferation. It is uncertain if this hypothalamic inactivity is secondary to her head trauma following a car accident, but the possibility exists. As far as her oligomenorrhea is concerned, she is making enough estrogen that she will have withdrawal bleeding to cyclic progestin and recommendations are that she take Provera, 10 mg. on the first 10 days of each month and she should expect to have a withdrawal bleed a few days following the cessation of this medication. If she does not have a withdrawal bleed with her cyclic Provera, she is informed that she needs to consider taking hormone replacement therapy in the form of Premarin 0.625 mg. on days 1 through 25 and Provera, 10 mg. on days 16 through 25. This patient is very disinclined to take estrogen since she feels it's what made her breasts grow and become asymmetric. On review of all parameters and even though the presentation doesn't make a perfectly full diagnosis, the diagnosis is most consistent with hypothalamic amenorrhea. MRI of the head to rule out any possible CNS lesions that might be effecting her cycling system was negative.

The patient was seen by a plastic surgeon, and after counseling, she elected a reduction mammoplasty.

This case is an unusual presentation and represents most likely a hypothalamic amenorrhea secondary to head trauma, and subsequent development of iatrogenic breast asymmetry.

The opinions expressed in this report are those of the author and do not necessarily reflect the views of the USPHS or USCG.

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(1) Commander, United States Public Health Service, Flight Surgeon, U.S. Coast Guard Air Station, P. O. Box 2542, Sitka, Alaska 99835.

Sexually Speaking . . .

Mary Cavalier, M.S.⁽¹⁾

The Dance

CASE STUDY

A gentleman named Michael, age 55, and his partner, present with low desire/low arousal. The partner is very upset. She has feelings of being rejected, unloved, and unattractive. Although Michael insists that she is lovable and attractive, he "just doesn't have any sexual feelings."

He has been on Catapres® for high blood pressure for several years. The couple was unaware that a potential side effect is impotence and/or decreased libido. He agrees to get with his doctor on the possibility of getting off the medication to determine if it is a side effect. The doctor is agreeable and the patient stops the medication. Several weeks later, no change in the libido has occurred.

DISCUSSION

The key factor in this study is the fact that he has been on the medication for a period of time. This is not to indicate that the side effects would take longer to disappear but rather that a "dance of intimacy" has been established between the couple which is actually at the core of the dysfunction. Let me explain further. . .

The dance of intimacy is very subtle. A glace across the room for one person may be an invitation for sex but is received as a mere passing look by the partner. The tricky part is that the non responsiveness may be interpreted as rejection. Hurt feelings and anger begin building the walls.

In the case presented above, low libido may indeed have been related to the medication in the beginning, but now after years of a subtle dance which has lead to hurt feelings and a lot of anger, the libido remains turned off.

TREATMENT

A couple must be willing to forgive the past and rewrite the music that they dance to. This requires a delicate look at the present dance in order for the couple to understand where the break down of communication occurred. It requires each person to be willing to risk rejection in order for the dance to be changed. What often sabotages the treatment is the pains of the past which create the fears of the future and paralyzes the couple in the same pattern.

As in anything, the couple must communicate clearly what they want. It is not uncommon for the couple to experience back sliding into the old behavior. This back sliding can be interpreted as an opportunity to dissect the dance and attempt a change. The key is that the couple must be willing to forgive and move on. A renewal of the commitment and recall of the foundation of the relationship is helpful.

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(1) Robert Alberts, M.D. & Associates, 3340 Providence Drive, Anchorage, Alaska 99508.

American Society for Circumpolar Health

TENTH INTERNATIONAL CONGRESS ON CIRCUMPOLAR HEALTH
Anchorage, Alaska
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GLIMPSES OF ALASKAN MEDICAL HISTORY

Edited by Robert Fortuine, M.D.

Syphilis control in Russian America - 1 (1845)

The medical articles written by Dr. Romanovskii and published after his return to Russia covered the period May 1, 1843, through May 1, 1845, and were originally extracted from official reports submitted to Governor A. A. Etholen. They described not only the diseases prevalent among the Russians, but those which seemed to affect the Alaska Native population selectively.

The reports also portray some of the details of Russian medical practice in the mid-nineteenth century. Romanovskii's therapeutic preferences were probably fairly typical of his time, with considerable emphasis on the use of bleeding, botanical drugs, and external applications. Bleeding was usually performed with a lancet, especially since leeches, as he complained, were in short supply in the colonies. Internal drugs he used included anti-inflammatories, emetics, laxatives, narcotics (poppy heads), and sudorifics. Externally, he employed aromatic stimulant poultices (including elder and camomile), hot salt and mustard foot baths, counterirritants, various ointments, and Spanish fly, a favorite blistering agent of the period. He was also a staunch advocate of smallpox vaccination.

Much of the text of his second report deals with control measures for syphilis in the colonies, at that time a widespread and worrisome problem for both Russians and Natives. Originally introduced from the outside, the disease had been prevalent for decades and seemed to maintain its vigor because of the frequent sexual liaisons between the Russian workers and the local Native population. Romanovskii described, as seen in the excerpts below, some of the measures which were undertaken—rather successfully, in fact—to control the spread of the disease. He followed this by a perceptive analysis in which he listed seven cultural, social, political, and economic reasons why such a control program could not be entirely effective. He concluded his report on syphilis by describing in detail some of the principal clinical characteristics of the disease as it manifested itself in the colonies.

"The appearance of syphilis on the Northwest Coast of America, must in all probability be ascribed to the first visits of American vessels to this coast. Their crews, infected on the islands of the Pacific, where this disease was very prevalent, then spread it here through relations with native women. . . . In 1822 the colonial leadership permitted the natives to settle . . . next to the wall of the fort in order to more easily observe their behaviour and foil harmful designs. . . . These natives, 700 in number, have constant relations with the Russians, in both trade and other matters, and along with supplying the settlement with necessary provisions, also transfer syphilis. Some of the native women, living in the forest all year, abandon themselves there to harlotry with the European employees in Novo-Arkhangel'sk, and thus infect many of them with syphilis.

"In order to protect people from such infection, the Medical Administration proposed various means. Of these the only one which seemed practical was forcible treatment of native women, which was effected for the first time, in 1842. By order of the Chief Manager of the colonies and after preliminary negotiations with the Toens [elders] several syphilitic women were then taken from the forest and sent to the fort for treatment. This was repeated annually for the next two years, especially in the current year, 1845. During the year, at various times, more than 70 women were taken into the fort of whom 33, appearing infected, were treated by me in the Novo-Arkhangel'sk womens' hospital and released as cured.

"The stated measures, indicating the care and attention given by the colonial administration to both the European employees in our colonies and to the natives, serve, however, only [to] reduce the number who have syphilis; one cannot eliminate it entirely. . . ."

"As for other sections of the Russian-American colonies, syphilis existed there in former times (both on the Aleutian Chain, inhabited by the Aleuts proper, and on Kad'iak Island, inhabited by the Kad'iaktsy), but at present neither I nor medical apprentices serving there have noted it anywhere. It was probably brought to the Aleutian Islands by the first Russian arrivals who settled on these islands. Before 1840 syphilis was found mainly on Kad'iak Island. In that year, as the result of a report by the Kad'iak Office about an increase in the number afflicted with this malady, I was sent there and upon arrival took the most active measures for its extirpation. All the inhabitants of Kad'iak Island were examined, and those showing the disease were hospitalized for treatment, until they were cured. Since then no more syphilis has come to light on this island."

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From Out of the Past. . . Over Thirty Years Ago

by Gloria K. Park, M.D.

Samuel J. Call, M.D. - 1858 - 1909

Reference - the Alaska Journal, Vol. 4, No. 3, 1974, article by Albert K. Cocke

Dr. Samuel J. Call clad for the rescue expedition to Point Barrow.

Samuel Johnson Call was born in Missouri in 1858, grew up in California and received his M.D. degree from Cooper Medical College in San Francisco. He also received training at the Post Graduate Medical School in New York. In 1880 he was hired by the Alaska Commercial Company (headquartered in San Francisco) and went to Unalaska as company surgeon. He regularly visited villages from Attu to Saint Michael. He left Unalaska in 1890 and in 1891 joined the Revenue Cutter Service, sailing on the "Bear" as her surgeon. Also aboard was Dr. Sheldon Jackson who was General Agent of Education for Alaska as well as a Presbyterian missionary. In addition to medical duties, Dr. Call was often involved in rounding up reindeer which were being moved from Siberia to Unalaska and then to Port Clarence. He provided medical care to the coastal villages, whalers and fishing vessels. The "Bear" sailed north each spring and spent several months in the Bering Sea and the Arctic.

Dr. Call received national attention attention when he served on the "Overland Expedition for the Relief of the Whalers in the Arctic Ocean" 1897-98. word had been received from Point Barrow that eight whalers were locked in the ice and did not have enough food to last the winter. The "Bear" had just returned from duty and was equipped and manned by volunteers and sailed within three weeks. The plan was to drive a herd of reindeers to Point Barrow for the whalers. They tried for Cape Nome but had to land at Tanunak due to heavy ice and Dr. Call was with the party of four to go overland and gather reindeer on the way to Cape Nome where they acquired 138 animals. they gathered at Cape Prince of Wales where W.T. Lopp joined them with a large heard of deer. They left with 448 reindeer and covered 700 miles in 55 days to reach Point Barrow with 382 animals. With the aid of Dr. Call and fresh meat nearly all the whaling men recovered. [In 1902 Dr. Call was awarded

Revenue Cutter BEAR north of Point Barrow, 1897.

The congressional gold medal awarded to Dr. Call in 1902 for his part in the overland relief expedition, 1897-98.

a "Congressional Gold Medal" for his part in the relief expedition.]

In 1899 Dr. Call became seriously ill and was put ashore at Saint Michael to recover. Gold had been discovered on the beach near the Snake River and a tent city arose, called Nome. Dr. Call resigned from the cutter service August 1899 and headed for Nome. There was plenty to do - measles, typhoid, pneumonia, smallpox, poor sanitation and other diseases caused much illness in the 25,000 population along the beach and streams. Dr. Call was appointed Quarantine Officer by the Marine Hospital Service in 1900 and later became City Health Officer for $250.00 per month. Dr. Call was in the news again in 1901 when he made a 600 mile overland winter trip in the winter to escort a violently insne priest to the mission at Holy Cross. It was a difficult and dangerous trip involving a blizzard and dodging open water on the return to Nome for a total of 1200 miles that took two months.

In 1902 Nome newspapers frequently reported that Dr. Call was in poor health and in August he left Nome on the steamer "Ohio". In 1903 he again joined the evenue Cutter Service and served on the "Thetis" for $150.00 per month. He later received the rank of First Lieutenant and was assigned to the Cutter McCullock on the Pacific. However his health was poor and he retired in 1908. He died at Hollister, California in 1909. His medical work in Alaska covered 25 years thus probably making him the first long term physician in Alaska.

Photos courtesy of the Call Collection, University of Alaska Archives.

Book Review

"HUMAN EVOLUTIONARY BIOLOGY"

A new book entitled "Human Evolutionary Biology" authored by Arndt Von Hipple, M.D. and published by Stone Age Press, Anchorage, Alaska.

New readers will enjoy the refreshing conversational style in which this scientific treatise is presented. Sir Francis Bacon was the last person who successfully learned all there was to know in the written English language. Now the audacity and arrogance of our friend and colleague, Arndt, have enabled him to do much the same thing in the area of medical science. His style is witty, charming, and personal. It sounds as though these are his personal secrets that he is passing on to the reader. This enjoyable book is both accurate as well as fun to read. Arndt's wit and clever turn of phrase is put to good use in this new endeavor. I heartily recommend the readers of ***Alaska Medicine*** to purchase a copy.

Clinton B. Lillibridge, M.D.
Pediatric Gastroenterologist

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Letter to the Editor . . .

PAINLESS HERPES ZOSTER

Dear Editor,

Case Report #1: A 23-year-old white male cook, active duty military, presents with a midline to midline unilateral blistery rash on the trunk. Diagnosis is shingles. He complains of no pain. He relates that six members of his family in Oregon had shingles in the past without pain. This information was historically, medically startling. He is a healthy individual, who works out at the gymnasium regularly, and after his convalescence knocked another individual out in a recent boxing match. He reviewed the family history at my insistence, and relates that his relatives had no discomfort from the shingles. I followed his case closely, discussed it daily, and he never once had pain. He works everyday. All his laboratory work-up for immunodeficiency was done thoroughly and has been absolutely negative.

Case Report #2: A 26-year-old white male aircrew, flight mechanic, reports a blistery rash midline to midline involving the ear and neck. He is uncomfortable, but not in agony. He is absolutely healthy, but reports possible exposure to a baby with chicken pox. One of my assistants wishes to try Zovirax® orally as reported by infectious disease specialists. Treatment is instituted by the book, and he heals without complaints. A full laboratory work-up is done, and is absolutely negative. There is no sign of immunodeficiency, immunosuppression, or anergy, or any positive findings. He is absolutely healthy, and works everyday without any complaints.

DISCUSSION

Frankly, I was timid about discussing my experiences until I read *Biomedical Inquiry*, Summer 1995, a pamphlet from my medical school, the University of Texas Medical Branch at Galveston. The front cover read "Curious Cuttlefish, Virus Express, Parents with Aids," and on page 3 - "A Painful Rash of Ignorance." It was my training through the years where I formed the impression that shingles was a bad omen. I remember my teachers telling me to look down the road after a shingles case for malignancy. I was taught that there was a higher incidence- question mark- of malignancy in patients who had shingles. Then, I ran across my painless shingles case in a normal person, and uneventful shingles case in another brawny aircrew above normal- type person.

Obviously, there are special situations occurring in the "virus field." Recently I was doing a search by Medline Grateful Med on "leukopenia," and suddenly, it listed a reference on the Oropouche virus in Brazil. I perked up because I was interested in the effects of benzene on the bone marrow, and leukopenia can be an early sign of toxicity in my human research observations, from the epidemiological standpoint. Referring back to the *Biomedical Inquiry* pamphlet, the first featured article was "Travels with Oropouche - Shipping hundreds of viruses more than a thousand miles is a delicate business." The article boasted slightly how UTMB, my medical school, had acquired a celebrated virus collection and equally celebrated Yale virologists. I immediately jumped back to page 3 to check out carefully that painful rash of ignorance. In my experience, I have two cases of not so painful a blistery rash, and I decided to dissect it out. I am no Lewis Thomas, and I wish he was here to check all of these latest developments. The painful rash of ignorance says: 1) one-fifth of us will get shingles — herpes zoster; 2) shingles is caused by the same virus that causes chicken pox; 3) the virus hides out in the nerve roots near the spinal cord before turning "painful and ugly". I always had a problem with this hiding-out-bandit situation, but I accepted it. Perhaps lesions in the spinal ganglia of the spinal cord nerve roots were reported long ago, and are still described and pathophysiologically factual; 4) shingles is not difficult to diagnose. It mentions the elderly as typical patients with a blistery rash after days or weeks of pain; 5) shingles is an emergency. It is a nerve infection. If it is not treated early, nerve damage can be irreversible, as well as the accompanying pain. This information was dramatic, and I was definitely paying attention. Shingles pain is much worse than kidney stone pain! 6) a study of 36 medical centers, 419 patients, in North America and Australia. Right there, I though of my Australian friends that I met in Sitka, Alaska, from the Esplanade, Coffin Bay, South Australia. I wanted to go there so badly, now I was a little unsure. Australians have the same problems that we have. The study revealed that patients taking famciclovir in the 72 hours after the first shingles blisters appeared, and on the drug three times a day for a week healed in about a month. The patients healed on the effect of the drug a few days sooner than a placebo group. I wondered there for a millisecond about "the power" in statistical research. Then, the article said that drug recipients lost the pain after an average 60 days, and that in the controls, pain lasted 163 days. Alright, the Lewis Thomas in you is asking right now, how can one explain a case of painless shingles with painless shingles relatives, and shingles in a brawny, healthy aircrew flight mechanic. I immediately flashed back to the days when as a USPHS trainee, I heard one of our professors discuss cases of Kaposi Sarcoma emanating from the French Quarter in New Orleans. He described the gross findings, and further discussed the pronunciation, as unlike the spelling, Kaposhi. I was very glad always, always to wear gloves. He mentioned further, how the final word was not out on Kaposi Sarcoma. Later, with the Aids epidemic, one could reflect on the wisdom of his words.

I must admit that when my patient with "painless shingles" got well, I fired off a letter to the authors of the *Textbook of Dermatology* by Moschella and Hurley. I looked up the closest address I could from the available early page, and wrote to the Lahey Clinic in Boston, and Philadelphia. I went to the post office and tried to get the closest zip codes I could find. Bother letters returned with a little hand pointed to "wrong address," "return to sender." That will teach them, not to spell out their addresses properly! I didn't have the courage to pursue it. I was frozen with my cases, my experiences. Then, I read A Painful Rash of Ignorance. Maybe, my medical school taught me correctly. Maybe, I should express my findings. Obviously, the professor who will depict his research in the Annals of Internal Medicine this summer, does not know about my painless shingles case. My former professor in New Orleans has no idea that I recalled what he said about Kaposi Sarcoma years ago, in his professorial wisdom. Any my friend, professor par excellence of Epidemiology and Biostatistics, does not know I recalled wondering about "the power" in a study, "statistical power," and its application in evaluating discomfort, pain and suffering. Sometimes we all get even, how about my "painless" shingles case, and not so remarkable shingles, flight mechanic. I admit I miss Lewis Thomas, and others like him- from a biological standpoint. I am proud to read Biomedical Inquiry, by UTMB-Galveston. Perhaps, all is not lost. Lewis Thomas passed away, and so did Jonas Salk, but we are still here, more average, still mortal men to help others. We are good disciples. "Painless shingles", for once, we all get even! With patience and time, time will take care of everything!

Manuel A. Rodriguez, M.D.
Cdr, USPHS, FS, USCG
Flight Surgeon, USCG
P.O. Box 2542
Sitka, Alaska 99835

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(continued from page 103)

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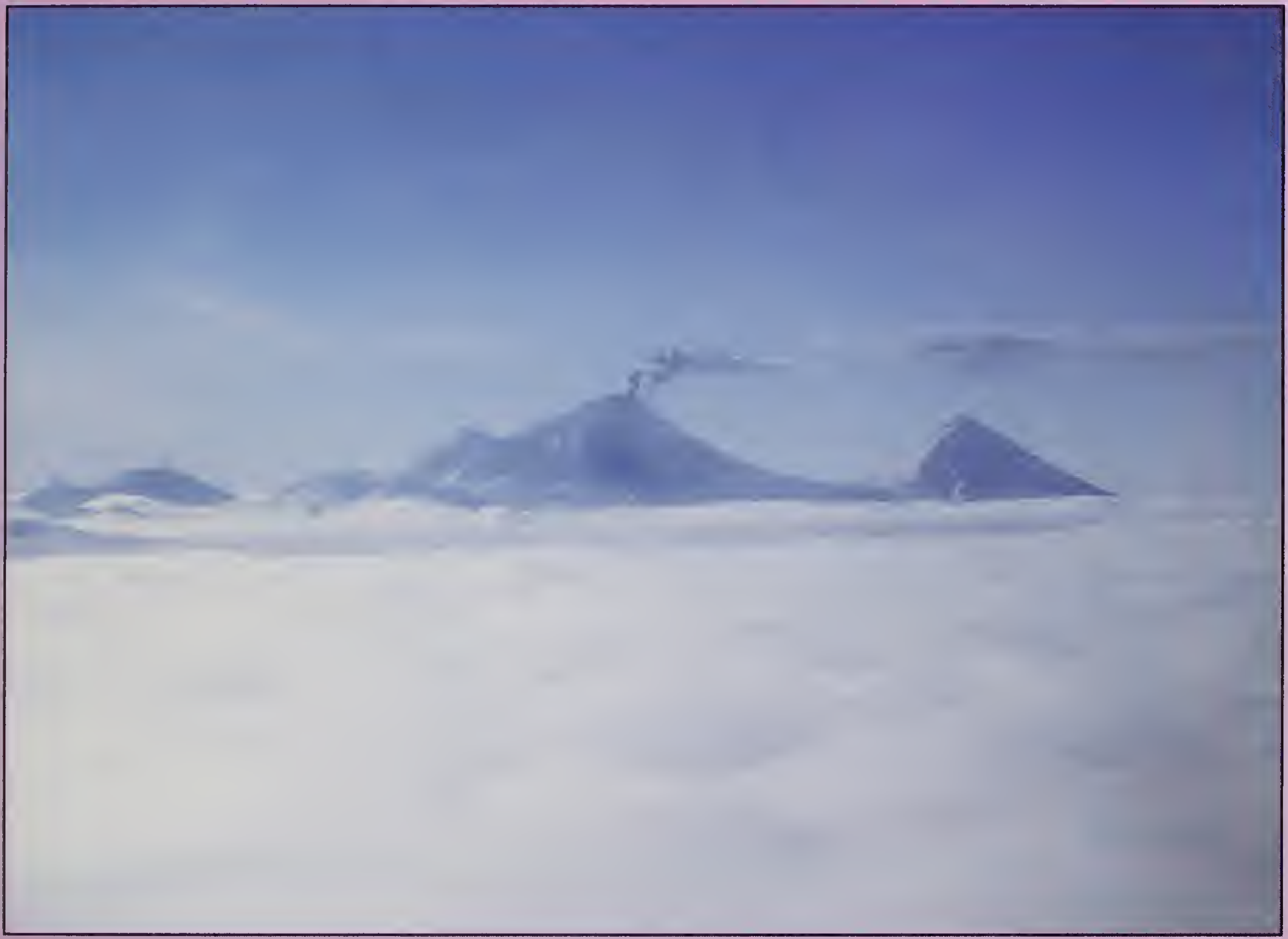

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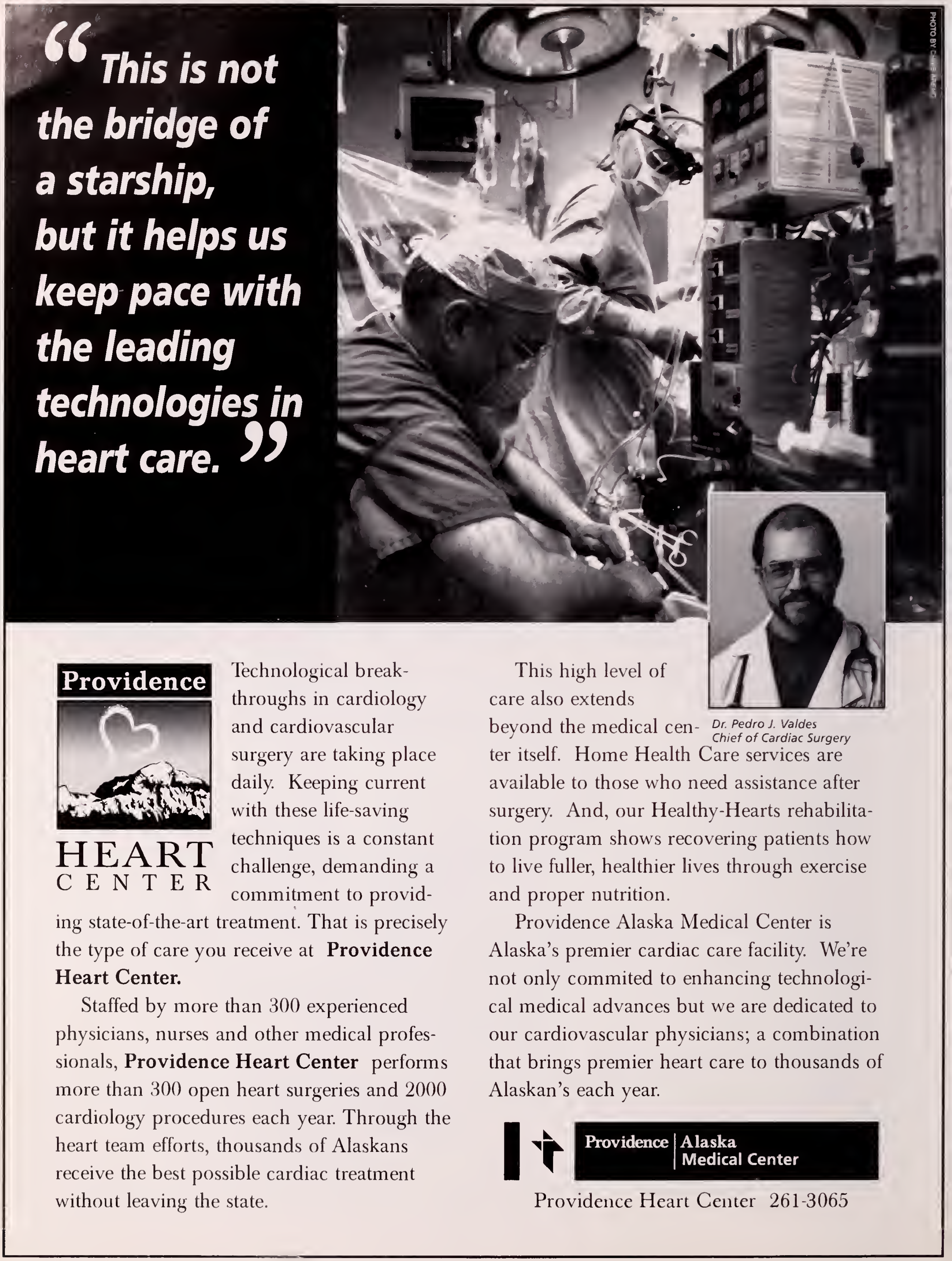
About the cover: Mt. Pavlov, Alaska Peninsula. Mt. Pavlov during Strombolian eruption, 1960. Photo courtesy of Donald R. Rogers, M.D., Anchorage, Alaska.



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YEARS OF POTENTIAL LIFE LOST AND LOST FUTURE PRODUCTIVITY

DUE TO OCCUPATIONAL FATALITIES - ALASKA, 1990-1994

Michael L. Klatt, MS(1)
Richard D. Kennedy, MS(1)
George A. Conway, MD, MPH(1)

ABSTRACT

Introduction: **Alaska had the highest occupational fatality rate of any state for the 1980s. The impact of these events is estimated by the index of years of potential life lost before age 65 (YPLL), which was developed to measure the potentially preventable mortality occurring early in life.**

Methods: **Lost future productivity (wages) and YPLL were calculated from surveillance statistics for all workers killed on the job during this 5-year period.**

Results: **During 1990-1994, Alaska experienced 343 work-related deaths among civilians under age 65. YPLL was 9,690 years with an estimated lost future productivity of $367,000,000.**

Discussion: **Premature death due to occupational traumatic injury in Alaska for 1990-1994 was extremely costly to society. Premature death not only adversely affects the deceased workers' family, friends, and coworkers, but also society economically. Effective intervention strategies are needed to significantly reduce both the number and the cost of fatal occupational trauma in Alaska.**

INTRODUCTION

The National Traumatic Occupational Fatalities (NTOF) surveillance system data for the 10-year period 1980-1989 showed that Alaska had the highest occupational fatality rate of all states, with 34.8 worker deaths per 100,000 workers, almost five times the U.S. rate of 7.0/100,000 for the same decade (1). Alaska's high occupational fatality rate was due to an inordinate number of job-related deaths in the industries of commercial fishing, air transport, and logging (2,3).

In addition to the devastating effect of a worker's death on family, friends, and colleagues, the social and economic consequences of each premature death reinforce the need to prevent death before its natural occurrence, so that individuals can contribute maximally to society(4). The negative impact of occupational fatalities on society can be quantified in terms of years of potential life lost before age 65 (YPLL) and lost future productivity.

In the U.S., conventional death statistics (crude and age-adjusted mortality data) are dominated by deaths in the elderly (5-8). The YPLL index was originally developed to measure the potentially preventable mortality occurring early in life (5-9). Today YPLL is also used as an impact measure to quantify societal loss from premature death (4). The major strengths of YPLL are that it is simple to compute and comprehend and that it focuses on areas where prevention strategies are needed (4-7).

It is also vital to translate the burden imposed upon society by occupational fatalities into economic terms to facilitate the decision-making process for social policy (10). Lost future productivity is defined as the dollar value of wages not earned by workers who die prematurely (10).

Analyzing YPLL and lost future productivity by industry provides a method to target those workers and occupations most in need of public health intervention (4). After identifying the worker populations vulnerable to premature death, YPLL and lost future productivity can be used to monitor temporal trends and to measure progress toward amelioration (3).

METHODS

Occupational fatalities in Alaska for 1990-1994 were identified via a surveillance system that included notification by governmental agencies (i.e., Alaska Department of Health and Social Services, Alaska Department of Labor, National Transportation Safety Board, Occupational Safety and Health Administration, and United States Coast Guard) and examination of Anchorage's daily newspapers.

---

(1) National Institute for Occupational Safety and Health, Division of Safety Research, Alaska Activity, 4230 University Drive, Suite 310, Anchorage, Alaska 99508.

YPLL was calculated by subtracting each worker's age at death from 65 for all 1990-1994 Alaskan occupational fatalities. The same method was used to calculate YPLL for the three Alaskan industries that historically recorded the greatest number of annual fatalities and the highest annual fatality rates: commercial fishing, air transport, and logging (1).

The estimated lost future productivity for workers killed on the job in the industries of commercial fishing, air transport, and logging were calculated by multiplying the YPLL for each of these three industries times that industry's mean annual income for 1991. The estimated lost future productivity for other worker fatalities was calculated by multiplying their YPLL times the 1991 mean annual income for all Alaskan workers.

RESULTS

During the 5-year period 1990-1994, 343* civilian work-related deaths with known ages under 65 years occurred in Alaska. Of these 343 occupational fatalities, 181 (52.8%) occurred in three industries: commercial fishing 117, air transport 44, and logging 20 (Figure 1). The mean age at death for these workers killed on the job in Alaska during this time period was 37.1 years. Of the three highest-risk industries, commercial fishing had the youngest mean age at death, 32.8 years, logging was next, 37.8 years, and air transport the oldest, 39.9 years. YPLL due to all occupational fatalities in Alaska for 1990-1994 was 9,690 years (Figure 2). Commercial fishing had the highest total YPLL with 3,764 years (38.8%), followed by air transport with 1,105 years (11.4%), and logging with 545 years (5.6%) (Figure 3).

Figure 1: Number of Deaths in Alaskan Workers, 1990--1994

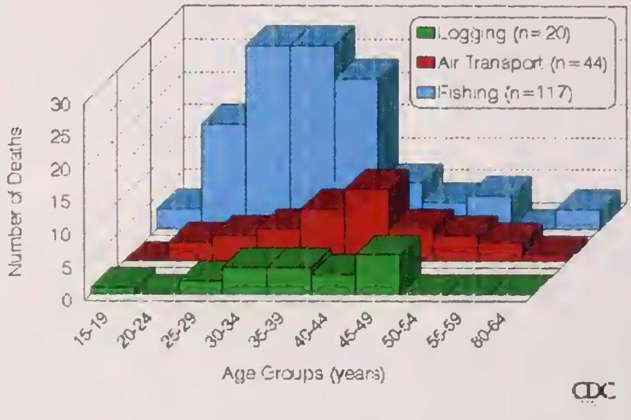


Figure 2: Years of Potential Life Lost (YPLL) per Occupation, Alaska, 1990--1994 (YPLL=9,690)

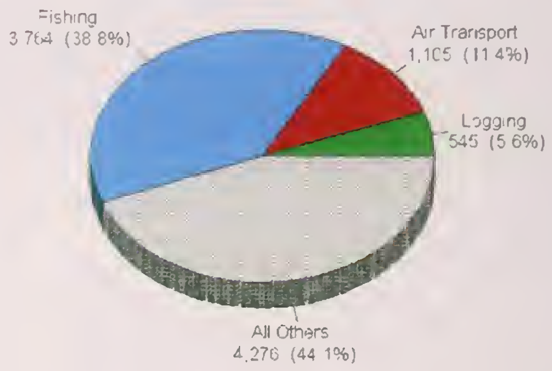
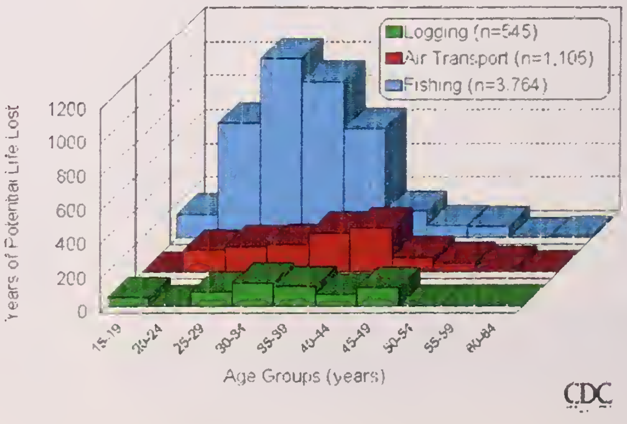


Figure 3: Years of Potential Life Lost (YPLL) by Alaskan Workers, 1990--1994



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*This number does not include 4 civilian workers over the age of 65 or 18 military personnel killed on the job in Alaska during this same time frame.

The mean annual income for all Alaskan workers for 1991 was $30,500 (11). The mean annual incomes for the same year for commercial fishing, logging and air transport were $46,500, $41,800 and $34,700 respectively (11). We estimate the lost future productivity due to these occupational fatalities in Alaska for 1990-1994 using the 1991 mean annual incomes to be $367,000,000. The Alaskan industries with the greatest estimated economic burden on society in terms of future wages lost for this 5-year period were commercial fishing $175,000,000, air transport $38,000,000 and logging $23,000,000.

DISCUSSION

Premature death due to occupational traumatic injury in Alaska for the 5-year period, 1990-1994 was extremely costly to society. The cost of these occupational fatalities to society was not only devastating to the lives of the deceased workers' families, friends and coworkers, but also in terms of the economic loss from lost future productivity. Additional monetary expenses to society as a result of workers being killed on the job that are not addressed in this article include, but are not limited to, public assistance for the deceased worker's family, higher insurance premiums, higher product prices, property loss/replacement, and medical and nonmedical care for the fatally injured.

Because the number of worker deaths in the Alaskan commercial fishing industry comprised the largest share of the total occupational deaths and the largest share of the occupational deaths in younger age groups, and because commercial fishermen had a much higher-than-average annual income, commercial fishing fatalities exacted the largest toll on society in terms of both years of potential life lost before age 65 and of lost future productivity. For these reasons the primary focus of intervention efforts to prevent occupational fatalities in Alaska should be on the commercial fishing industry, followed by air transport and logging.

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Alaska Department of Fish and Game
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Alaska Marine Safety Education Association
Alaska State Troopers
Federal Aviation Administration
Federal Occupational Safety and Health Administration
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National Transportation Safety Board
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Cancer Home Health Care

Introduction

As part of the National Cancer Institute's effort to advance knowledge on the costs of cancer and the economic burden of continuing care for cancer patients and their families, the Institute held a conference on home care for cancer patients. Preliminary research in preparation for the conference revealed trends and patterns in the delivery of homecare in the U.S. Home health and personal health care were the fastest growing of all health expenditures between 1960 and 1993.(1) Three sets of government programs provide home care services to Americans: public insurance (eg., Medicare and Medicaid), the Veterans Administration and the Indian Health Service.

The papers that follow focus on cancer homecare for American Indians and cancer care delivery through Indian Health Service Community Health Representatives. They present what is known about cancer incidence, survival and mortality among American Indians, describe the Community Health Representative (CHR) Program, and explain some clinical concerns related to cancer care among American Indians. These contributions demonstrate the difficulties of providing cancer care to an impoverished population with scarce resources; however, they also make clear that culturally sensitive providers who can share knowledge about programs and funding options are key to efficiently managing scarce resources as well as helping families assure quality care for cancer patients.

As these articles show, CHR programs have woven safety nets of health care for American Indians in vicinities with them by providing medical and nursing services, health, education, training and other services, such as transportation, to help cancer patients gain access to medical and nursing services at home or in clinics and hospitals.

Successful cancer care requires the sophisticated management of a team of health care providers. Articles by Steele and Hisnanick and Kramer suggest that trained personnel in the CHR program have been able to manage the care of cancer patients at relatively little cost. Yet, the essays by Kaur and Burhanstappanov reveal that serious problems in appropriate cancer care delivery remain. For example, IHS facilities are scarce (which unduly burdens CHRs who provide transportation to facilities) and oncologists in Indian country are rare. In addition, inadequate numbers of American Indians are sampled in surveys, and even the best data sources, including the SEER cancer registry, collect information on only a few tribes even though tribes vary considerably in health status and cancer incidence. Finally, census misclassification likely leads to an undercount of American Indians, which results in unreliable denominators for cancer rates. In short, programs serving American Indian cancer patients, including the CHR program, need accurate data collection to evaluate cancer care among the American Indian population.

While the conference produced many fine articles, these contributions are especially provocative because they reveal a little-known system already in place in the U.S., the IHS CHR program, which may provide a model for low-cost, high quality home care delivery. By focusing on a population with relatively few resources, these studies show the full range of services critical to successful cancer care. Ongoing management of the range of medical and nursing services, including transportation, information and training are all necessary components in comprehensive cancer care delivery. By managing the medical burden of cancer, a sound cancer care delivery program enables families to focus on nurturing the patient and strengthening family relationships during the crisis engendered by cancer.

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Home Health Care for Cancer Patients: Insights from the American Indian Community

Lois Steele, M.D. (1)
John Hisnanick, Ph.D. (2)

ABSTRACT

With the confounding effects of an aging population, as well as advances in the treatment of cancers, the cure rates and associated recovery time for patients have increased. In the past, recovery from cancer treatment was often associated with extended hospital stays. However, shorter lengths of stay and the use of home health care is now the accepted standard of care. For the recovering patients, and their families, sensitivity to their cultural needs may often be overlooked given the diverse nature and background of home health care providers.

Within the Indian Health Service, the cultural needs of the patients are taken into account. Through this paper we hope to provide a description of this unique program of home health care for American Indians who live on or near reservations and trust lands and Alaskan Natives who live in traditional villages.

INTRODUCTION

The objective of the study is to provide a description of the provision of home health care for cancer patients in the American Indian (AI) population who live on or near reservations, given the existence of various cultural norms within these communities. Such a presentation is important because this sub-population is often ignored in traditional research on the subject. For the purposes of our discussion, home health care will refer to the provision of a combination of both medical care and social services for those individuals inflicted with a chronic or debilitating condition due to cancer (1). The site of the care for these patients is either their home or that of a family member.

In general, the AI population does not face the same issues and concerns regarding access to care as do other minorities. Care is provided, by public law, through the Indian Health Service (IHS) (2). However, there are complex eligibility requirements on service area, tribal enrollment status, as well as the time and reasons the patient is off the reservation, that determine what care is available.

The IHS furnishes comprehensive "womb to tomb" care to eligible American Indians and Alaska Natives (AI/AN) without regard to socioeconomic status, age, or state of residence. In this respect, the agency's role is quite different from other federally run or legislated health care programs, such as Medicare/Medicaid, Department of Veterans Affairs, and Department of Defense. Within these programs, health care services are dependent upon age, income eligibility requirements and prior or current enlistment in a branch of the armed forces. Another distinguishing characteristic of the IHS is the broad range of services that it provides. The IHS provides not only primary care and preventive care but also environmental health services and home health care. Primary and preventive health services are provided through a health network composed of forty-two hospitals, sixty-five health centers, four school health centers, and fifty-two health stations. For the approximately 51% of the population that does not live on or near reservations, some health care can be obtained through urban clinics. These facilities tend to be extensively staffed by members of the United States (US) Public Health Service. However, the provision of home health care by the IHS tries to incorporate the cultural diversity that makes up its service population. This is achieved through the Community Health Representative (CHR) program, whose employees are hired from the local community and are usually tribal members.

Even more diverse than the services provided by the IHS is the population which they serve. There are currently 512 federally recognized tribal entities within the US, accounting for under two million in total population. Slightly under half of all the American Indians live on reservations. This population is more rural and younger than is the US general population (3). In addition, they experience an unemployment rate 1.6 times greater than does the general population and AI households are also 2.3 times more likely to be at or below the poverty level (4).

---

(1) Medical Research Officer, U.S. Dept. of Health and Human Services, Indian Health Service, Office of Health Programs Research and Development, Tucson, AZ.

(2) Economist, U.S. Dept. of Veterans Affairs, National Center for Veterans Analysis & Statistics, Biometrics Division, Washington, DC. Formerly with the Indian Health Service.

CANCER AND THE AMERICAN INDIAN/ ALASKAN NATIVE EXPERIENCE

At the turn of the century, cancer was a rare disorder for American Indians and Alaska Natives (5). When compared with other groups in the U.S. for 1989, the cancer mortality rate (83 deaths per 100,000 population per annum) still seems low. The age-adjusted cancer mortality rate for AIs is only 0.6 that of the total US population. Nevertheless, over the past twenty years cancer rates have increased significantly in the AI population. It is now the third leading cause of death for all ages and the second leading cause of death for AIs over age forty-five. Cancer is the second leading cause of death in AI women and the third leading cause of death in men(6).

AI/AN have the poorest survival rate of any racial group for all sites of cancer. Good survival data has not been available very long for AIs, but what is known is based largely upon the work done using the New Mexico Tumor Registry (SEER) data (7,8). In 1977-83 AI women had a low reported incidence of breast cancer of 21 per 100,000, but a five-year survival rate (1975-84) of only 46 percent. By comparison, the incidence of breast cancer among black women was 73 per 100,000 with a five-year survival rate of 63% and among white women the reported incidence was 91 per 100,000 with a five-year survival rate of 76%.

A similar outcome is observed when looking at the incidence and five-year survival rate for men and prostate cancer. Among AI males, the reported incidence from 1977-83 was 31 per 100,000, with a survival rate of 54%. By comparison, among black males the incidence of prostate cancer was 119 per 100,000 with a five-year survival rate of 62% and among white males, an incidence of 64 per 100,000 and a survival rate of 61%(9).

The New Mexico Tumor Registry (SEER) data also indicates that the survival rates for members of southwestern tribes are the poorest of any racial group in the US, even when the stage at diagnosis and treatment is taken into account (8). Inter-tribal comparisons of survival rates are not possible, given the lack of published data associated with other non-southwestern tribes. In addition, there is underreporting of cancer statistics within the urban Indian population (7).

Although the reported incidence of cancer among AI/AN is relatively low, the burden of cancer, often associated with lower survival rates, appears to be much greater. Lower survival rates have both social and economic implications such as the burden imposed upon individuals and their families due to the deterioration in the patient's quality of life, as well as the economic implications associated with premature death (10). Therefore, given the cultural, geographic, and genetic diversity of the 512 AI/AN tribes, relieving some of this burden through home health care requires both community involvement and assessment. For example, all treatment and control efforts toward cancer must recognize the important role of traditional healers, as well as the spiritual beliefs of community members.

CANCER AND THE AI/AN COMMUNITY: CULTURAL INSIGHTS

The strong spiritual and cultural beliefs of many traditional AI/AN are important considerations when looking at home health care and cancer patients. The provision of home care for cancer patients in the AI/AN population can be problematic given the cultural uniqueness and tribal customs that are present in these communities. For example, in one specific southwestern tribe (11), if a person dies in his or her own traditional home or that of a family member, the dwelling cannot be used again. The dwelling will be destroyed and a new one built. If the family is not as traditional and the dwelling is more modern, there may still have to be many traditional sings (or ceremonies) before the family is comfortable living there again. In either case, these views must be accounted for in the delivery of home health care. To handle such problematic issues, both informal and formal structures of care exist. The informal structure is the help and support provided by family, friends and other tribal and community members. The formal structure of care is the Congressionally mandated CHR program.

Informal Structures of Care:

As a way to highlight the informal structure of care that exists within this culturally diverse population, consider the following account of the burden of cancer on a urban, southwestern AI family. This account is not represented as common to all AI/AN families, but is used to provide an insight into how one family, with strong spiritual and cultural ties, faced this situation.

The traditional Indian way often involves the telling of a story (12). Victor, an AI teenager, lived in an urban, southwestern city. Like other teenage boys, he was an athlete, playing noseguard on his high school football team. He was a popular young man, with a sensitivity and love toward others and a spiritual insight. At age fifteen he was diagnosed with lymphoblastic leukemia. After his diagnosis, it was important to him to be at home as much as possible during the eighteen months between the time of diagnosis and his death. At home there were various types of support from the family, friends and tribal members. Victor's family is not the nuclear family often referred to in the popular media. His family, as well as those of many other AI families, consisted of four generations of people who all shared in the burden of his care. The extended family, which included his siblings, nephews and nieces, were never shielded from the reality of his illness. From the time of diagnosis, the entire family was involved in his illness. Many of his providers felt that the exceptional love and support the family gave Victor was rooted in their strong tribal beliefs.

In addition, the support given was the source of Victor's inner strength. Every Monday evening after he was diagnosed, he fasted and left a plate of food or sweet grass outside as an offering to his ancestors. He seemed to have a philosophy that the illness was a burden he had to bear. He stated, "When my time comes, my ancestors from the spirit world will come for me." The spiritual leader of the Tribe explained, that in ancient times, death was not feared; it was welcomed. It meant rebirth forever in heaven (13).

Eight years after Victor's death, one regret of his parents was that he could not have been home more during those eighteen months after the diagnosis was made. He preferred home to the hospital. Yet, the hospital was where he had to go for therapy and care. His family recalls the many trips to and from, and hours at the hospital. Victor's family is proud that they were able to handle these expenses themselves and did not have to use resources of the spiritual leaders or deprive others who did not have large families, to help. Siblings, aunts and uncles as well as godparents contributed towards the family's expenses, (i.e., gas and food money, rides to and from the hospital, time with the patient and even money to buy the special food and supplies the patient needed).

Some tribes have a system of helping tribal members if they do not have a family. A natural outgrowth of the extended family system which is present in various forms in different tribes, is the IHS Community Health Representative Program; the formal structure of care in AI/AN communities.

Formal Structure of Care:

The CHR program is an extended provider, community-based program established by Congressional mandate in 1968 that became fully operational by 1972. The program's broad objective is to provide a community health outreach component to meet both community and tribal-specific health care needs. The emphasis is on health promotion and disease prevention activities in these communities and on meeting the needs of rural tribal members who cannot always travel to the IHS facilities(14). Care is provided by on-the-job trained, community-based workers.

The official mission and definition of the CHR program is well documented and presented elsewhere (15), but the program's real success is based upon its providers of care. These providers are from the local community, are usually tribal members, and are usually proficient in their tribal language. Training for these providers is gained through an intensive three-week course designed and implemented by the IHS(see appendix A) as well as on-the-job experience.

The CHRs work closely with public health nurses. In fiscal year 1990, CHRs recorded 47,021 visits to patients with cancer. Although this represents only about 1% of the total number of visits made to AI/AN, the numbers do not adequately reflect the real impact these providers play in the lives of their patients. Their role is to enhance and supplement patient care by providing such services as translation, but they often coordinate, arrange and supervise services provided through other federal, state or tribal programs. For example, the Bureau of Indian Affairs funds and operates a "Homemaker" program. This program provides patients with needed housework help as well as with activities of daily living during the week. Such visits are often coordinated by the CHRs to occur during their regular program visits. In addition, CHRs will provide medically necessary transportation.

The CHR program varies from tribe to tribe, yet the mission remains the same. By using local people, whom cancer patients often know and trust, the CHR program provides a formal mechanism for the delivery

Table 1.

Relevant Program Information Concerning Appropriations, Number of CHRs and Number of Patient Encounters Covering Years 1986-1991.

	1986	1987	1988	1989	1990	1991
Appropriations*	$25.0	$25.0	$26.3	$28.0	$29.6	$36.5
% change		0.0	5.0	6.48	6.04	23.3
# CHRs	1473	1410	1411	1363	1432	1512
% change		-4.28	0.07	-3.4	5.06	5.59
# Patient Encounters*	N/A	N/A	N/A	N/A	4.03	4.26
% change						5.56

Note: * - implies values are represented in (approximate) millions.
N/A - implies data not available.

of informal services. Specifically, the provision of home health care in rural settings.

Lastly, the CHR program can be viewed as at least one governmental entity that achieves efficient delivery of services, given budget constraints. For example, in fiscal year 1992 the average cost per CHR provider for the lower forty-eight states was $22,600; for Alaska it was slightly higher, $28,300. This yearly figure includes salaries and fringe benefits, travel and per diem, as well as training costs and materials. In addition (Table 1), in fiscal year 1991, CHRs had over 4.2 million client contacts, at an estimated cost of $8.50 per visit (16).

DISCUSSION AND FUTURE RESEARCH

Home health care has become and will continue to be an important component in the provision of health care, especially for cancer patients. Even though significant strides are being made in reducing cancer mortality through early detection and better treatment, recovery still takes time and support. Given that the general population is aging, living longer and experiencing shorter lengths of stay at hospitals, there is a need to investigate other options for expanding the provision of home health care for cancer patients.

There are existing programs that provide home health care for cancer patients in the US. However, a problem with home health care services under the present configuration is that programs are often fragmented across various agencies, with respite care under one agency's responsibility, hospice care the responsibility of another, and nursing care under still another. Some of the services provided are duplicated and others are underutilized. Within urban areas, patient care services are often aided by the coordinating efforts of such service organizations as the United Way, but in rural areas, few, if any, home health services are available for cancer patients. This raises questions regarding the equitable provision of home health care and whether a paradigm exists for the efficient use of resources associated with care giving.

The IHS Community Health Representative program plays an integral part in the provision of home health care for cancer patients in AI/AN communities. This is a unique, federally funded program that could provide insight into questions related to the equitable and efficient delivery of home health care for cancer patients. In addition, the CHR program appears to be a cost effective model that could be used as a starting point in building comprehensive federally funded home health programs for cancer patients.

In pursuing future research on issues dealing with home health care and cancer patients, one advantage that the CHR program provides is an existing data base with uniform standards and procedures for recording patient encounters (16). This information can provide the basis for investigating both general and specific issues dealing with the equitable and efficient provision of home health care for cancer patients and for home health care in general (17).

Lastly, the previous discussion highlighted how this program and its data base can be used for future investigations, but it may never get at the true measure of worth of the CHR program. That is, to quantify the associated benefits of a small subsidy that will allow everyone to care for their loved ones at home.

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The opinion and views expressed are those of the authors and no way reflect those of either the Indian Health Service or the U.S. Dept. of Veterans Affairs

APPENDIX A

Outline Of Basic Training Curriculum For Community Health Representatives

I. ORIENTATION

II. COMMUNITY HEALTH
  a. The CHR Program and the Role of the CHR
  b. Community Health Concepts
  c. Contracts and Grants
  d. Field Trip

III. COMMUNICATIONS
  a. Introduction to Communications and Active Listening
  b. Health Education
  c. Planning and Presenting a Talk

IV. THE BODY IN HEALTH AND DISEASE
  a. Disease Concepts
  b. Anatomy and Function of Body Systems
  c. The Body's Response to Disease
  d. Vital Signs
  e. Client Visits: Recording and Reporting
  f. Nutrition in Health and Disease
  g. Disease with Community Impact: Diabetes II
  h. Dental Health
  I. Genetic Factors

V. FAMILY HEALTH
  a. Maternal/Child Health
  b. Family Planning
  c. Sexually-Transmitted Disease

VI. ENVIRONMENTAL HEALTH
  a. Prevention of Water and Waste-borne Diseases
  b. Prevention of Insect and Animal-borne Diseases
  c. Prevention of Food-borne Diseases
  d. Injury Control
  e. Transnational Environmental Concerns

VII. Mental Health
  a. Mental Health Concepts
  b. Major Mental Health Problems
  c. Mental Health Crises and Intervention
  d. Alcohol Abuse
  e. Substance Abuse and Chemical Dependency
  f. Dysfunctional Families

Home Care for Native American Cancer Patients: An Oncologist's Perspective

GUEST EDITORIAL

Successful care of cancer patients today requires a sophisticated team effort. The recent improvement in survival of a variety of cancers is due to combined therapy, often including surgery, radiation, and chemotherapy. When the therapy of cancer is aggressive with the intent to cure, intensive supportive care including home care is required. Today most radiation and chemotherapy regimens are delivered on an outpatient basis. Reservation-based Native American patients in particular may be forced to travel great distances for state-of-the-art cancer care. For example, it is not unusual for some of my patients to travel 400 miles round trip for treatment and evaluation!

While overall cancer survivor statistics in the U.S. show that over fifty percent of patients diagnosed with cancer today will be cured, those rates are documented to be significantly lower for minority populations (1). While cancer was once said to be quite rare for Native Americans it has recently been increasing dramatically. It is now the second leading cause of death among American Indian and Alaska Native women and third among men in those populations (2).

The SEER data showed that Native American survival rates from cancer are the poorest of any minority population studied (3). If this grim toll is to be corrected we must make progress on several fronts including prevention, screening and supportive care. As a community oncologist, I find the supportive care provided by home care services invaluable for patients. Without that care many patients would prematurely stop their cancer treatment due to side effects or die of complications such as infection. For patients who are not curable it also makes a major difference in terms of maximal quality of life such as pain control. For example, ambulatory infusion pumps allow for home administration of morphine for terminal care. Periodic nursing supervision is essential to such a terminal care program.

The Indian Health Service (IHS) is a bureau of the Health Resources Services Administration, an agency of the Public Health Service. It was founded in 1955 by a transfer of health services from the Bureau of Indian Affairs, Department of Interior. Since that time IHS has grown larger and more complicated as a national organization responsibile for direct and contract health care services to approximately one million people. Through commitment to preventive health programs substantial progress has been achieved in improving the health of American Indians and Alaska Natives. While Indian Health Service nurses and other providers have traditionally provided limited home care services, it is time to evaluate these services in relation to national trends, agency policies and the burgeoning need.

There are many incentives for planning appropriate home care services to American Indians and Alaska Native cancer patients, which include:

1. Improved quality of life for the patient
2. Reduced hospital length of stay
3. Reduced total hospitalizations
4. Reduced transportation costs for travel vouchers
5. Improved hospice services to terminal patients

One major stumbling block to implementing more home care services is the lack of medicare funding to IHS patients. Because of the current wording of the medicare and medicaid sections of the Social Security Act, Title XVIII and XIX funds from those programs are available to hsopitals and skilled nursing facilities run by IHS but not to home care agencies (4). Unless coverage is expanded to include home health care, it will be difficult with constant resource allocation limits for IHS to provide the pesonnel and technology needed. Currently, many families make great sacrifices to try to care for the cancer patient at home and to bring them great distances for evaluation.

There is also a lack of accurate data on the numbers of Native Americans who suffer from cancer. Each data base has limitations when trying to look specifically at American Indian and Alaska Natives and as a result multiple sources must be reviewed to assess the cancer problem among indigenous peoples. Such data desperately needs to be refined so that long term strategic planning, including availability of home care services, can take place to lower the morbidity and mortality from cancer in this special population.

Judith Salmon Kaur, M.D. (Choctaw/Cherokee)
Medical Oncologist
Mayo Comprehensive Cancer Center
Rochester, MN

(references on page 156)

Native American Data Limitations and Home Care

Linda Burhansstipanov, MSPH, DrPH⁽¹⁾

Dr. Judith Kaur prepared an editorial for this journal on "Home Care for Native American Cancer Patients" because of her well-acknowledged expertise, and rapport with indigenous patients. One of two American Indian oncologists in the U.S., she has actively worked with hospice care. She has provided exemplary cancer patient support services and is highly regarded for her skill, sincerity, and depth of understanding among Indigenous Peoples.

"Cancer" carries multiple cultural barriers and the support of one's family and community assists in coping and ideally, overcoming the disease. Some Indigenous communities utilize home care while others ostracize the cancer patient for fear that the disease will spread to others. There are no data to clarify how common home care is among indigenous communities. Dr. Kaur's editorial refers to data problems.

Based upon suggestions from the reviewers of Dr. Kaur's editorial, this paper was written to elaborate on the following issues which were introduced in her editorial: (1) travel to cancer services; (2) limitations of national surveys; and (3) limitations of national cancer databases.

TRAVEL TO CANCER SERVICES

Indigenous cancer patients rarely have access to services from the local area. The majority of patients travel great distances to oncologists who are contracted to the Indian Health Service. Although the Service will pay for travel, it is economically prohibited from paying for the transportation of a family member or loved one to accompany the patient. The cancer patient frequently must face the cultural sterility of Western medicine as well as the trauma of a cancer diagnosis and treatment alone. The travel costs are prohibitive for Peoples who are subsisting at poverty level. For example, a cancer patient from St. Paul Island (one of the Priblof Islands in the Bering Sea) must fly for over 2 hours to Anchorage for treatment. The cost for this flight is over $700. Because of the weather, the Alaska Native is unable to utilize boats for much of the year. Likewise, the cancer patient in Montana travels to Seattle for cancer treatment. The travel problem is not limited to remote rural regions. An American Indian living in the Washington, D.C. area must travel 400 miles to Cherokee, North Carolina which is the nearest IHS facility.

Community Health Representatives (CHR) are Indigenous People who are trained by the Indian Health Service (IHS) to provide outreach services. As one of many diverse duties, CHRs in most IHS Areas provide free roundtrip transportation for patients between their homes and the IHS medical facility. However, the number of available CHRs may be insufficient to provide travel services to indigenous patients. For example, there is one CHR assigned to cover approximately 60,000 American Indians living in the Greater Los Angeles Basin. L.A. has a very poor to non-existent public transportation system and reliance on CHR transportation may be the only means to get to the American Indian Clinic in Bellflower.

LIMITATIONS OF NATIONAL SURVEY DATA

The National Health Interview Survey (NHIS)

The National Center for Health Statistics (NCHS) is responsible for administering the National Health Interview Survey (NHIS) annually, which the Center uses to collect information on the health of civilian non-institutionalized Americans. The NHIS provides data on the incidence of acute illness and injuries, the prevalence of chronic conditions and impairments, the extent of disability, and the utilization of health care services by persons seventeen years of age and older. Data are collected during a 20-90 minute personal interview on a cross-sectional sample of households that are representative of the U.S. population, but not of individual states. Census interviewers collect information on approximately 46,500 households and 122,000 persons and generally maintain an annual response rate of 95 percent.

The two-part NHIS questionnaire includes a set of basic health and demographic items that are administered annually, and a supplemental survey of current health topics which includes questions from several sources outside of NCHS. In the 1987 and 1992 Cancer Supplements, the National Cancer Institute submitted a section on cancer-related risk factors and an epidemiologic study questionnaire. The latter includes specific questions related to diet, screening examinations, and family history of cancer.

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(1) (Western Cherokee) Director, AMC Native American Cancer Research Program, Denver, CO.

Population surveys conducted by NCHS have collected ample information on Caucasians, blacks, and Hispanics to begin to understand the variety of health issues affecting these populations. Since 1985, blacks have been over-sampled to improve the precision of estimates for the population. Hispanics were over-sampled for the 1992 survey. The Hispanic Health and Nutrition Examination Survey (HHANES,1982-84) also included a section on cancer risks and collected information on several epidemiologic questions.

There have been insufficient numbers of Native Americans responding to the NHIS to draw any conclusions. For example, there were only 149 out of 22,043 (0.006 percent) American Indian and Alaska Native respondents 18 years and older who completed the Cancer Control Supplement in 1987 and 135 out of 22,080 (0.006 percent) American Indian and Alaska Native respondents in the Cancer Epidemiology Supplement. In 1990, there were 288 out of 41,104 (0.007 percent) American Indian and Alaska Native respondents included in the Health Promotion Disease Prevention Supplement (NCHS). These compare quite unfavorably with American Indian and Alaska Native representation in the U.S. population, which in 1980 was 1.4 million (0.6 percent) and in 1990 was 1.9 million (0.8 percent) (1).

Survey of American Indians and Alaska Natives (SAIAN)

In 1987, a Survey of American Indians and Alaska Natives (SAIAN) was conducted using the National Medical Expenditure Survey Instrument. During the American Public Health Association meeting in 1991, the validity and reliability of these results were challenged. For example, a Yupik elder asked if any Alaska Native people had been included in the survey, since she had lived her whole life in the region described by the SAIAN and didn't know anyone who acted as described in the results. The American Indian and Alaska Native people who were in attendance felt that it was obvious that the survey information was interpreted by non-Native investigators who were limited by their lack of cultural understanding. For example, a Navajo data collector could have recorded a phrase that has a specific connotation among Navajo People, but was interpreted differently by a non-Native researcher. Some parts of this survey are likely to be accurate and of assistance in developing cancer programs. The survey sample and methodology were well designed, but the results of the study have been met with skepticism by American Indian and Alaska Native People.

The major concerns are that (1) the survey instrument was not adapted for cultural appropriateness nor competence, especially those for whom English was a second language; (2) interviewers translated or interpreted survey questions for the participant and introduced interviewer bias; and (3) the responses, which are likely to have multiple connotations among Native and non-Native cultures, were interpreted and analyzed by non-Natives.

LIMITATIONS OF NATIONAL CANCER DATABASES

Policy makers, researchers, and health care professionals at federal, state and local levels rely on national databases as accurate, representative sources of information. However, the U.S. Census underestimates the number of American Indians and Alaska Natives in the U.S. which results in denominator problems. The National Cancer Institute's SEER Registry data are accurate for American Indians living in Arizona and New Mexico, but those data cannot be extended to Indigenous Peoples living in other regions of the country. The National Center for Health Statistics receives inaccurate racial classification data from the States and as a result underestimates the number of Indigenous Peoples who die of cancer. The Indian Health Service databases are limited to the 33 Reservation States and include about 25% of the Native population. The IHS databases are racially accurate but have ICD coding errors.

DISCUSSION OF DATABASE LIMITATIONS

Close examination of each database reveals problems such as racial misclassification, undercounting, or use of small segments or regions of the country which are not representative of Indigenous Peoples living in other regions (Table 1). As a result there is no single national database which accurately describes the cancer problem among Indigenous Peoples. Multiple databases must be relied upon to assess the cancer problem among American Indians and Alaska Natives. The heterogeneity of these sovereign nations and their lifestyles has contributed to difficulty in drawing conclusions about the reported lower incidence of cancer as compared to other minority groups.

The types of data which are available for U.S. whites and blacks are unavailable for the majority of Native Americans. What little such data are available are accurate for Indigenous Peoples living in the states of Arizona and New Mexico. Based upon the limited data available, cancer incidence appears to be increasing throughout Indian Country, and is worse in Alaska and the Northern States. Accurate statistics are critical for epidemiologic research and for the design,

Table 1.

Summary: Purposes and Limitations of National Cancer Databases Related to American Indians and Alaska Natives[9]

DATABASE	PURPOSE OF DATABASE	LIMITATIONS
U.S. Census	To identify descriptive demographic information for all population units	• Undercount of Native Americans • Racial misclassification • Insensitive to annual fluctuations Effects of limitations: affects accuracy of the data used as the denominator to generate rates, underestimates the number of American Indians and Alaska Natives and overcounts other racial groups
Mortality Data (National Center for Health Statistics)	To identify the frequency of deaths by cause (based upon death certificate information provided by each state)	• Racial misclassification Effects of limitation: undercounts of numerator data is likely to have greater impact in underestimating incidence and mortality rates in American Indian and Alaska Native populations with lesser impact of overestimating rates in the general population.
Surveillance, Epidemiology, and End Results (NCI SEER Program)	To collect cancer incidence, and survival data on a routine basis from designated population-based cancer registries in various areas of the country; to analyze trends in cancer incidence, mortality (based on NCHS data), and patient survival in the United States	• Racial misclassification • Most complete data are for Southwestern Indian tribes Effects of limitations: affects the accuracy of the numerator by underestimating the number of American Indians and Alaska Natives; Southwestern American Indian data are NOT generalizable to Indigenous Peoples from other regions of U.S.
Indian Health Service	To calculate IHS "user population" IHS patient care rates; IHS "user population" figures are used in calculating vital event rates since state birth and death certificates do not provide information on use of IHS services.[10]	• Quality control related to medical records, e.g., data entry and coding of diagnosis • Limited to 33 reservations states • Partial and/or lack of urban American Indian and Alaska Native cancer data • Limited to its "user" population (i.e., used a facility within the last three years) Effect of Limitations: Data represent small portion of reservation American Indians and not the majority of the Indigenous populations.

implementation, and evaluation of public health interventions (2).

The most frequently cited data are from the U.S. Census. The accuracy and comprehensiveness of data collected for the U.S. Census directly affects the quality of the "denominator" which is used in Native American statistics. In spite of U.S. Census efforts to include Indigenous Peoples in the 1990 Census, data from many Native Americans were not included (e.g., absent due to job migration during the months of Census collection; refusal to participate as a protest against providing additional data to U.S. government). The 1980 Census estimated a 6% undercount of Native Americans and the estimate is at least as high for the 1990 Census. Among the several problems with the Census questions is that the race question allows Indigenous Peoples to identify their "ancestry" rather than the of degree of Indian blood. There are likely to be many errors in self-identifying data. Although Indigenous groups claim that there is an undercount on the U.S. Census, Hahn (1992) states that there is an overcount of American Indians when self-identity or ancestry are used in Census data (3). For example, in the 1980 Census, 6.8 million persons reported American Indian ancestry, but only 1.2 million reported American Indian race. However, members of the Senate Select Committee on Indian Affairs estimate (1992) that there continues to be an undercount of American Indians and Alaska Natives in the 1990 Census.

Reputable national cancer databases, such as the National Cancer Institute's Surveillance, Epidemiology, and End Results (SEER), collect cancer data on a routine basis from designated population-based cancer registries in various areas of the country. The New Mexico SEER represents a significant population of American Indians (19%) and the data are of excellent quality for American Indians living in Arizona and New Mexico. However, the tribes which are included in the New Mexico SEER have behavioral patterns which vary greatly with Indigenous Peoples from other parts of the country and therefore the data are not generalizable. For example, only 21% of the Indians in Arizona and New Mexico smoke cigarettes as compared to 70% of urban Indians in the northern Plains, or 56% of Seattle urban Indians, or 60-70% of Ojibwa Indians in Minnesota, or 65% of the Sioux.

SEER sites are located in other areas where there are known to have high numbers of American Indians, such as Seattle, Los Angeles, and San Francisco. However, racial misclassification is a severe problem (error ranges between 30-78% in those databases) in every area except for the New Mexico SEER Registry. Racial misclassification occurs as a result of the data collection and recording process. The funeral director completes the forms which identify the race of the deceased patient for mortality data; health personnel, such as the hospital intake worker, completes records which include racial identification for incidence and survival databases. These health professionals sometimes assume the race of the patient rather than asking the patient and/or the patient's family.

The quality of databases varies among racial groups and among geographic regions of the country. For example, the data from the New Mexico Tumor Registry are among the more comprehensive and accurate records of cancer among American Indians and Alaska Natives. New Mexico Tumor Registry staff recognized the need to collect accurate and precise data on all people, including underserved populations, such as American Indians and Hispanics. As a result, a data collection protocol was implemented which accurately ascertains the race and diagnosis of the cancer patient. Other tumor registries may not have had the same level of interest to incorporate similar techniques until recent years. Collection of accurate racial information is now a higher priority in tumor registries, such as the Fred Hutchinson Cancer Research Center in Seattle.

Seattle's racial misclassification is estimated to be approximately 30% ("born Indian, die white") (4). A recent (1993) review of data for the state of California provided a racial misclassification of 80% (2). Hahn (1993) examined race-specific rates of infant mortality from 1983-1985 in the thirty-three states which contain federally designated tribal reservations and found the consistent recording of "American Indian" or "Alaska Native" on birth and death record to be 52.1% (2). Frost (1992) examined the racial misclassification of the NCI Puget Sound SEER cancer registry to determine the extent to which racial misclassification contributed to the observed low cancer incidence among Native Americans. The misclassification ranged from 11% for full bloods to 52% for Natives who have blood quantum less than 50% (5). Sugarman examined death certificates to determine if misclassification of Indian race on death certificates result in underestimation of the true burden of cancer mortality among urban American Indians in Seattle. Twenty-nine percent of indigenous patients were racially misclassified and of those, 68% were coded as white on their death certificates (6). In 1991-1992, the racial misclassification error rates were estimated to range from 15 to 35% in mortality databases in states such as Washington, Montana, Wisconsin, and Minnesota. Seventy-five percent of American Indians and Alaska Natives living in Wyoming were racially misclassified on death certificates in the State Cancer Registry (7).

Indian Health Service (IHS) databases include diagnostic and ICD coding errors such as recording an ICD code for cancer prior to receiving laboratory confirmation of the diagnosis, or listing an ICD cancer code for a disease which is not cancer. These coding errors are estimated to be 11% and may result in overcounting cancer incidence (8).

The IHS administration and Director of Cancer Prevention and Control has expressed significant concern regarding these errors and IHS Area Databases are being reviewed to correct the coding and/or diagnostic errors. The correction and improvement of IHS Area databases has been identified as a priority within the IHS Cancer Prevention and Control Program and as of 1992, steps have already been implemented to initiate those corrections.

The IHS databases are primarily composed of approximately 25% of the American Indian and Alaska Native population. These databases are limited to 33 of the 50 states, only include Indigenous Peoples who have utilized the services within the last 2 years, and do not include Indigenous Peoples who utilize third party payment for their health care. Due to budgetary and personnel constraints, little can be done at this time to overcome these limitations.

Mortality data are recorded at the local level, submitted to the state and then subsequently to national mortality databases, such as the National Center for Health Statistics and the National Death Index. However, local data records contain errors which result in undercounting the number of actual cancer deaths. The primary problem is racial misclassification which was previously described.

FEDERAL AGENCIES' RECOGNITION OF THE DATA AND INSTRUMENT PROBLEMS

During the last few years, federal agencies have sponsored meetings with and/or reports for Native Americans regarding data quality issues. The consensus from these meetings and reports is that national data bases do not provide adequate American Indian and Alaska Native data. For example, federal agencies have sponsored seven meetings during 1992 which included review of database problems such as racial misclassification. Although there is a clear consensus from these and other meetings of the need for the development of culturally competent and appropriate survey instruments and materials, there are no established guidelines or protocols for determining the cultural appropriateness of an instrument.

RECOMMENDATIONS

States need to revise the data collection procedures presently utilized to reduce the racial misclassification errors in national databases. Since the New Mexico SEER Registry has a training program available which has resulted in accurate databases, other states may wish to utilize a similar type of training program in their local areas.

In addition, urban Indian clinics, native owned and operated health clinics (93-638 settings), and IHS facilities need to collect current and accurate information regarding their local service populations and utilize a common protocol for collecting and recording such data (e.g., the accurate recording of ICD codes following laboratory confirmation of diagnoses).

There is a dearth of data for American Indians and Alaska Natives which is necessary for the development, implementation and assessment of innovative cancer prevention and control research interventions. These racial data need to be collected based upon:

- Geographic regions, e.g., Alaska or the Northern Plains where cancer rates are significantly higher than for Native Peoples from other regions.
- Native communities exposed to cancer risks which are known to exist for non-Natives, e.g., Pine Ridge Reservation Indians subsisting on high fat commodities as their sole source of food.
- State tumor registries, particularly in states with a large number or proportion of American Indians and Alaska Natives.
- Isolated Native communities in which the gene-pool has limited introduction of non-Native genes (9).

Tribal and Native community-specific data need to be collected by the local American Indian and Alaska Native groups rather than by federal agencies because:

- Lack of federal staff, protocols and equipment to collect, record and transfer such data to a national database.
- Total cost is prohibitive (considering effort versus outcome).
- Difficulties in accessing American Indians and Alaska Natives who live in urban areas unless they utilize one of the 34 urban Indian clinics.
- Difficulties in collecting information on American Indians and Alaska Natives who utilize third party payment.
- Cancer information is collected differently among urban Indian clinics, Indian-owned and operated clinics, and IHS facilities (e.g., many do not use ICD codes and many do not include the cancer site or stage at diagnosis).
- Some tribes are very small, and rate calculations are subject to instability based on small denominators.
- Difficulties in accessing some tribal communities because of tribal politics or migratory lifestyles.

Information is needed from the 500 plus federally recognized tribes, hundreds of state-recognized tribes, urban Indian clinics, American Indian owned and

(continued from page 137)

operated clinics and IHS facilities, and local Native communities. Data are essential to initiate efforts to accurately record cancer incidence, mortality, survival, and site data among Native Peoples. Federal and state governments may support local Native American organizations and communities in the development and implementation of culturally acceptable data collection techniques which utilize a common protocol for collecting and recording cancer data (e.g., the use of ICD codes).

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B. Josea Kramer, Ph.D.(1)

INTRODUCTION

No longer a rare occurrence, cancer is the third leading cause of death for American Indians of all ages and American Indians have the poorest five year survival rates of any population group in the United States (1). Home care, often a component in treatment plans, is a neglected area of health research for minorities. Home care utilization studies have focused on white, black and Hispanic populations and have neglected American Indians(2). This is particularly surprising because the American Indian family is an important resource for its members. In Indian Territory "to be poor is to be without family (3)." The role of the American Indian family in providing health care to its members is complex and may involve practical care, advice, and performance of religious rituals to supernaturally protect family health and well-being. Home care is an important health concern for American Indian communities. In one urban American Indian community, nearly one-fourth of elders are limited in their abilities to conduct Activities of Daily Living, suggesting that home care is not an unexpected consequence of the aging process (4). At this time, the Administration on Aging and the Indian Health Service are developing or examining the feasibility of programs to support home health care on reservations and American Indian colleges are training home care paraprofessionals to provide high quality home care for frail American Indian elders (5-8)

Home care links formal and informal systems of health care and is affected by social and cultural issues. These macro-level considerations provide a context for home care of cancer patients and are the focus of this article. Cultural beliefs and practices determine how individuals experience and express illness, treatment and recovery; influence the social organization for networks of support, self-care, and other health seeking behaviors. Treatment and adherence to treatment plans are affected by such social factors as access to health care systems, institutionalized racism, poverty, and the structure of social services.

MACRO-CULTURAL CONSIDERATIONS

Cultural diversity

American Indians are an extremely diverse and heterogeneous population composed of approximately 500 federally recognized nations, tribes, bands, and Alaskan Native villages, and an additional 100 non-recognized tribes in the United States. Today about 150 languages continue to be used.

Just as there is no single American Indian language, there is no single cultural tradition. The U.S. public is most familiar with northern Plains cultures, such as Lakota, thanks to Hollywood movies (e.g., "Dances with Wolves"), and with cultures which have become highly identified with their arts in a commercial market, such as Navajo weaving, Pueblo pottery, or Kwakiutl carvings.

The focus on material culture overlooks the startling contrasts of culture, society, and environment. For instance, the Pueblo theocratic agriculture-based societies of the Southwest are the oldest continually inhabited cities in North America. The cultures exploiting the Northwest Coastal areas were organized into socially and politically ranked village-based societies in which accumulation of surplus wealth was highly valued. In contrast, membership in a Plains Indian band was more fluid, reflecting both the egalitarian political structure, as well as the flexibility for individuals to select residence among the most favorable resource conditions.

Even within a geographically-defined Culture Area there is substantial tribal variation, evolving in response to social, technological, political, and economic dynamics within the region. For instance, "Indians of the Southwest" refers to societies which, at first contact with Americans, included sedentary agriculturists (e.g., Hopi, Pueblos), pastoralists (e.g., Navajo) and semi-nomadic hunter-gatherers (e.g., Apache).

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(1) Associate Director for Education and Evaluation
Geriatric Research Education and Clinical Center (GRECC)
Sepulveda Veterans Health Administration Medical Center

Rural - urban variation

Cross-cutting the cultural variation among American Indians and Alaskan Natives is the dispersal from indigenous small-scale communities into urban areas. Federal policies which encouraged relocation to industrial centers, particularly those following World War II, resulted in the division of American Indians into rural-reservation and urban populations (9,10). The rural-reservation population (i.e., those living on reservations, in trust territories, rural historic Indian territories in Oklahoma, and Alaskan villages) benefit both from their own sovereign political and cultural institutions and from Federal governmental institutions which are responsible for their health, education, and well-being. Much of the research and available data on American Indian health focuses on these concentrated populations. In contrast, urban American Indians are dispersed across the nation and throughout the general population (11) and are not represented by special governmental institutions.

Various waves of migration under changing federal policies contributed to the considerable regional variation in urban settings. Midwest and northeast cities are relatively homogeneous with respect to tribes, while the west coast cities are strikingly diverse (9). For instance, the 360 older American Indian respondents to a needs assessment survey in Los Angeles represented 92 tribal affiliations (4). Most (99%, n=231) Los Angeles elders (median age 58 years) were born on or near reservation or rancheria communities. Significant differences between tribal affiliation and health behaviors (e.g., cigarette use) and household size and structure indicate the persistence of normative culture patterns off reservation (12).

Tribal, regional, and individual variation

Cultural heterogeneity is associated with variation in health behaviors and in incidence of cancer. Smoking and lung cancer present a clear example. In the southwest, where cigarette smoking tends to occur at low rates, there are lower rates of lung cancer than have been noted among the tribes characterized by heavier smoking patterns on the northern Plains.

Occurrence of cancer appears to vary both among geographic culture areas and among cultural groups within a region. For instance, American Indian men living in Montana have higher incidence rates for kidney, lung, and prostate cancers than American Indian men living in the southwest. While rates of cervical cancer are elevated in American Indian women in Montana and in New Mexico, the incidence for breast and lung cancer is higher among the northern Plains than the southwestern women (13). There is also variation within a region. In Alaska, for example, cancer frequencies vary among the three indigenous populations of Eskimo, American Indian and Aleut (14).

Family and care-giving

In the absence of formal services, American Indian families provide many levels of care. American Indian care-givers, unlike their Anglo counterparts, may not express guilt and anger toward their dependent elders (15). The attitude of passive forbearance is not fatalism but an embracing acceptance of present-day reality.

Nevertheless elder urban American Indian care-recipients reported that neither community-based human services nor family home care was sufficient to meet their perceived need for assistance with Activities of Daily Living (4). Requesting additional assistance, however, may pose a dilemma for the elder if health-seeking behaviors, such as transportation to medical appointments, impose hardship and sacrifice of wages for another family member. Decisions about health care may be family-oriented rather than client-oriented. For community-based long term care services to be responsive to American Indian patients, the services should support the efforts and resources of the family.

The family support system is an essential component of treatment compliance. Unfortunately, the social service system is not always supportive of the Indian family, tending to provide financial assistance to conventional family roles such as daughters caring for older parents (16). In contrast, when the care-givers were unmarried grandsons providing transportation to dialysis, medical appointments or rehabilitation clinics, homemaking, cooking, and personal care to their grandparents, they were deemed ineligible for in-home supportive service stipends because, as young men, they should be in vocational training or in the work force. The "white tape" of bureaucracies can be a daunting challenge and a significant barrier to American Indian families.

Preferences and willingness to give or receive care from family members also reflects cultural diversity, even for the care of universally respected elders (17).

Generalizations about the American Indian family (18) tend to obscure variations of family structure which define the domestic division of labor and affect home care. In terms of family support, consider the difference between a matrilineal, matrilocal society, such as Navajo, and Euro-American societies which favor neolocal residence. In the latter, a married couple establishes an independent residence, reflecting economic and social resources. In a matrilocal, matrilineal society, a married couple establishes residence in the same or proximal households with the wife's female kin. Among reservation-dwelling Navajo, intergenerational reciprocity between matrilineally related women ensures care of frail grandmothers in the residential group (19). Older unmarried Navajo men, however, do not necessarily reside in their matrilineal residential group (e.g., in the camp of a mother, sister, or sister's daughter), may become isolated and at risk for early death (20). Such a skewing of risk is not associated with neolocality where either men or women can become isolated from kin during their lives.

End of life issues affecting home hospice care may reflect both tribal variation and degree of individual adherence to traditional norms. In the Southwest for instance, Navajo must avoid contact with the dead as a safeguard against ghost sickness and family members may be especially vulnerable. In another tribe, the dying family member must be attended so the body can be properly flexed for burial soon after death occurs. In a third tribe, the belief is that the soul departs the body three days after the body expires and therefore the corpse should not be immediately moved.

Health Beliefs

In general, traditional American Indian health care practices are holistic, do not distinguish separate models for mental and physical illnesses, and promote wellness. Indigenous systems of health care involve individuals in an assessment of their own state of health as well as active participation in healing. The notions of "silent disease," preventative medicine, and, to some extent, specialized medicine are foreign. Healing practices may be discrepant with the western medical model because they are based on culturally determined patterns rather than on medically relevant information related to the disease (21-24).

Uranium mining on the Navajo reservation exemplifies the relationship between an environmental health issue and socio-cultural factors. High rates of cancer are reported among Navajo who worked in the mines or lived nearby (25). This occupational and environmental health hazard was not mitigated by protective gear for workers or education of residents. Cancer is seen by many Navajo as a culturally valid outcome for disturbing the sacred nature of the land by strip mining. Therefore families have not aggressively pursued either treatment or legal options. The strong association between clans and their land base rules out voluntary relocation from the now environmentally hazardous zones. Navajo take the long view: in time the earth will heal itself and the health problems will likewise diminish.

Cultural values and health

Community-based American Indian health interventions often share the common approach of defining health beliefs and behaviors (and thus promotion and prevention strategies) in relation to the community rather than the individual (26). For instance, The American Indian Cancer Control Project in California rephrases smoking from a personal behavior to one that negatively impacts the health of the family and the community: The program slogan is "It's Your Life -- It's Our Future." (27)

Adherence to traditional values has been correlated with health-promoting behaviors (28) including rehabilitation. A Sioux home health nurse expressed her frustration in caring for a non-Indian client:

"Non-Indians really miss out by not having a tradition [like ours]. . . .She's 89 years old and she is angry that she is going to die of cancer. What hasn't she done yet in life? When you tell an Indian that they are going to die of cancer, they always question, 'Are you sure? How do you know?' Then they accept it and open a door on a new part of their lives."

The cultural value which this nurse expressed was to fully live in the here-and-now of present-day circumstances.

Traditional values can be successfully integrated into treatment plans for home care and rehabilitation (29). Some of the pertinent values include orientation to the present day, orientation to a past which honors tradition, oral transmission of knowledge, establishing a harmonic therapeutic relationship which reflects the value of harmony between individuals and society, and discrete communication which respects the individual. This holistic approach can, at times, be at odds with the western biomedical model's emphasis on mind-body dualism, individualism, and stratified relationship between the health care practitioner and the client.

MACRO-SOCIAL FACTORS

Access to Health Care

An important difference between the urban and rural/reservation populations is access to health care. Eligibility for medical care by Indian Health Service may vary depending on tribal affiliation and residence. Where Indian Health Service medical care is mandated and free, care may be linked exclusively to the reservation. Tribal members become ineligible for medical care after a period of 180 days residence off-reservation (30). (Eligibility is immediately reinstated when reservation residence is re-established). Since 1977 the Indian Health Service has allocated funds for urban clinics. The successful urban clinics have evolved with diversified funding sources (31) and may also serve non-Indian patients. The urban clinics tend to serve low income American Indian clients for whom wellness may be less immediate than survival issues. Regardless of location, long term care services, including home care, are not a priority under the current Indian Health Service funding allocations which are based on Years Productive Life Lost and which favor acute, prenatal, pediatric care.

In most urban areas with large American Indian populations, linkage to community-based long term care support services is limited at best (10). Urban American Indians are typically underserved by the federally funded network of aging services. The gap in services is caused by a combination of factors: allocation of services based on an economy of scale which penalizes small populations, access barriers, and lack of policy initiative. Typically, urban elders are also more disadvantaged than their reservation peers in receiving critical linkage information about psychological and supportive services from their attending physicians (32).

Consequences of poverty on health

Poverty, as a risk factor for cancer, puts many American Indian families at risk. Economic data from the 1980 US Census indicated that 27.5% of the American Indian population lived in poverty. In urban areas, poverty reduces access to health care. Poverty and lack of health care insurance diminishes the likelihood that individuals will pursue referrals or treatment options. A recent study of women's health at nine urban health clinics, found that one third of American Indian women were uninsured, further reducing their options to seek treatment specifically for cancer and other illnesses (33).

Racism and Stereotypes

U.S. society exhibits a systematic and racist bias against American Indians. Where it is not overt discrimination, American Indians are reduced to stereotypic caricatures (34). As an example, mainstream Americans apparently find it acceptable behavior to dress their children as Indian "princesses" or "braves," although it is no longer acceptable to dress children in blackface or as Chinamen with queues. Mainstream adults are still heard to indicate a "real Indian" to their children as if his/her humanity was not otherwise self-evident.

Institutionalized racism

On an institutional level, the Federal government banned the practice of Native American religion in the 19th Century and did not restore that right until the 1974 Indian Freedom of Religion Act. That legislation protects Indian *beliefs* but so far has failed to protect certain indigenous religious *practices*, such as the sacramental use of peyote by the Native American Church, access and possession of eagle feathers for ritual uses, and protection of sacred sites. Given that traditional American Indian health care practices are intimately tied to their systems of religion (3), failure to protect these health practices influences self-care and the willingness to use formal medical services. Perhaps it is not surprising when American Indians are reluctant to discuss alternative health treatments in a formal setting, although the systems of health care may be perceived as complementary.

Consequences of racism in health care settings

Lack of awareness about American Indians pervades health settings. First, American Indians report that they do not expect to be treated fairly by non-Indian health care and service providers (35) and prefer treatment by culturally sensitive providers, who are usually also Indian (36,37). The local American Indian Clinic's health educator notes that non-Indian providers may have a stereotype about Indian stoicism which, in the health care setting, reduces the likelihood of prescribing medications for pain relief, or reduces the likelihood that patients will be encouraged to share their feelings, concerns, and even symptoms.

Second, health care providers may not be knowledgeable about relating to the cultural needs of Indian patients (38). Indeed, they may fail to even recognize these patients. A recent examination of the Surveillance, Epidemiology, and End Results (SEER) cancer registry in Puget Sound indicated that the low incidence of reported cancers among American Indians is at least partially related to a 40% misclassification of race for Indian patients with a strong association ($p=<.001$) between blood quantum of 50% or less and misidentification (39).

Communication across stereotypes

Successful therapeutic relationships with an American Indian patient are not unique in the need for the health care provider to establish an interest in the patient's well-being. While busy health care professionals may feel comfortable "getting right down to business," American Indian patients may prefer to use the initial contact period to establish the knowledge, sincerity and trustworthiness of the provider (37,40). American Indian patients tend not to inform social or health services providers when they have been offended, instead they refuse services and do not return (15).

Non-Indians may find that listening can be a very demanding skill and requires a great deal of concentration, especially if the patient uses a formal oratory style, presenting the subject in different ways, using analogies, allegories, and examples. Accustomed to jumping right into a conversation, non-Indians may inadvertently interrupt the speaker — not too differently from an uneducated audience clapping between symphonic movements. Silence, lengthy pauses for consideration, and breathing are all parts of a communication style, and may not indicate hostility or discomfort. Depending on the individual and on his or her cultural background, there may be differences in etiquette between American Indian patients and non-Indian physicians which inhibit establishment of a therapeutic relationship (3,38). For instance, making eye contact may be a sign of frankness among non-Indians but is considered rude, an invasion of privacy, by American Indians. Contrariwise, the American Indian custom of politely avoiding eye contact may be wrongly interpreted as furtive. Health and human services need to be flexible and responsive to individual cues about appropriate behaviors.

A recent survey about cancer found that 98% of urban Indian women believe that annual PAP smears are important; however, a considerably smaller percentage reported having the PAP test done within the last year (70% for those under 40 years; and 55% for those over 40 years). One reason for this discrepancy is the American Indian patient's expectation that health care providers must order exams, whereas health care providers expect patients to make annual appointments for examinations (32). Mahoney (41) has suggested that, given the rising rates of cancer, health care providers must bear the responsibility to promote cancer screening and to ensure necessary follow-up.

DISCUSSION

The heterogeneity of this population and variation in cancer occurrence suggests that a single approach will not be effective to enhance home care. This review has highlighted cultural differences between tribes and social differences between reservation and urban populations. Just as health promotion campaigns should respond to local variation in cancer occurrence, effective strategies to improve home care should reflect local cultural and social factors. This suggests that community-based approaches will be the most productive strategy to implement national health campaigns to improve the status of American Indian cancer patients.

We know little about home care utilization by American Indians in general and by American Indian cancer patients in particular. Health care issues tend to focus on the concentrated reservation populations for whom Indian Health Service is mandated to provide care. Less is known about urban American Indians who are dispersed among the general population and represent a relatively small population, usually no more than 1% of a multi-ethnic community. A future research agenda should not only clarify home care utilization patterns but demonstrate best practice models and policies to marshall resources in support of home care for both rural and urban American Indian cancer patients.

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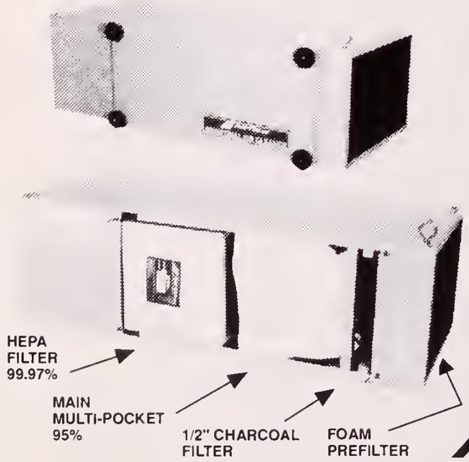

Group A Beta Hemolytic Streptococcus Pharyngitis in United States Coast Guard Units in Sitka

Manuel A. Rodriguez, M.D. [1]

The following report is a summary of two years surveillance of throat culture results for Group A Beta Hemolytic Streptococcus (GABHS) in an overseas location for Coast Guard unit members, dependents, and retirees. Patients were examined, cultured, treated, and recultured for therapeutic efficacy. Treatment consisted of Amoxicillin or Erythromycin for ten days. Cultures were processed by an independent quality controlled laboratory. The population reported comprises military families associated with the Air Station, Coast Guard cutter, Aid to Navigation, and Marine Safety Units in Sitka, Alaska. Computerized culture results were matched and filed by member identification numbers.

The table categorized patients by age, gender, and military designation. Active duty air station member - AS, cutter - C, navigation - N, safety - S, dependents - DAS, DC, DN, DS, and retirees R.

The data shows 46 positive cases of GABHS out of a total of 7,949 patients seen for the two year period recorded. Of the population, 0.01 percent was documented positive by culture technique. One active duty pilot from the air station was positive for GABHS. The patient had a maxillary sinus polyp resected later. All other cases of GABHS occurred in dependent members including mothers and offspring, and a dependent father. Twenty-seven dependents were from air station personnel, 17 were from cutter personal, one was from the Marine Safety Office. No retirees were positive for GABHS. No active duty members from the cutter navigation, or the Marine Safety Offfice were positive for GABHS. After review of the data and correlation of the positive cases to member distribution, positive cases were directly related to mothers and offspring in the same home environment. Positive cultures were more prominent in the dependents than in the active duty population. All patients revealed negative follow-up recultures except for a six-year-old boy who had a tonsillectomy, and by history following this review and transfer to a different military base, considered a carrier by specialist evaluation. The active duty population is considered healthier than the dependent population. This finding was correlated to less exposure to the home environment. The major home environment correlated to positive cultures was military housing. The only positive active duty member with GABHS ha(l maxillary sinus pathology. One adult male dependent with a home-based occupation was positive for GABHS. The active duty spouse was negative for GABHS. One out of 46 GABHS positive cases was an active duty helicopter pilot. The remainder were dependents of active duty personnel. The majority of GABHS positive dependents were from the home-based environment. The data shows that GABHS in this overseas location correlates with the dependents home-based environment, and is not a work-associated problem.

DISCUSSION

GABHS is manifested by fever, sore throat, and red exudative tonsillar pharyngitis in typical cases. Twenty percent of cases have this presentation, and the remainder are asymptomatic, or have fever, sore throat, or nonspecific symptoms. Precautions are to be observed to ensure that diagnosis is accurately made. Throat culture performance must be controlled by inoculation with a known strain of group A streptococcus.

A bacitracin disc test for presumptive diagnosis is reliable when a purified subculture is used. Cultures that are negative after 24 hours are reincubated an additional day before being discarded. Follow-up recultures after 10 days of appropriate therapy should be done. Ten percent of children with fever and sore throat have a streptococcal infection. If the patient or a family member have frequent streptococcal infections or if a history of rheumatic fever exists, a carrier state can be resolved by tonsillectomy if they continue to recur despite proper antibiotic prophylaxis. Ten to 15 percent of children in some studies are asymptomatic pharyngeal carriers of GABHS. In this study a 6-year-old male was evaluated by a specialist for the carrier state, and underwent tonsillectomy. His father had a history of rheumatic fever. Many studies have described the importance of infection transmission in occupations involving close contact between children and adults such as home-based child care, day care centers, and schools. GABHS is not

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(1) Commander, USPHS, FS, USCG Flight Surgeon, P.O. Box 2542, Sitka, AK 99835.

listed as a prominent representative of other work-associated environment exposures. In this review GABHS was correlated best to the dependents home-based environment, and not to the active duty work environment. Approximately 0.01 percent of all patients seen had GABHS. Two percent of all the GABHS cases consist of active duty members. The rest were dependents. GABHS was not considered a major problem in this study of USCG units in this overseas location for active duty members.

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Interstitial Pulmonary Fibrosis - Presentation in Aviation Medicine

Manuel A. Rodriguez, M.D. (1)

CASE HISTORY

The patient is a 33-year-old white male aircrew flight mechanic with several year history of intermittent hemoptysis. Chest x-rays were normal until three years ago when they began to show increased interstitial markings. A complete pulmonary work-up included a negative PPD, negative CT scan of the chest, negative fiberoptic bronchoscopy with brushings and biopsies. Past medical history consisted of asbestos exposure five years previously, an eight-year history of one pack per day cigarette habit, and owner of pet birds for the past three years including a parrot. He never lived in southern California, or the Ohio River Valley. He had no apparent chemical exposure, or allergies. His physical examination was essentially unremarkable. Pulmonary function studies showed an FVC of 5.63 (90%), FEV1 of 4.17 (82%). Repeat chest x-ray demonstrated increased interstitial markings. Room air blood gas revealed a pH of 7.41, pCO2 38, pO2 94. Coagulation studies and hematocrit were normal. The patient underwent a right lateral thoracotomy with an open biopsy of the right upper middle and lower lobes. All cultures including fungal, AFB, aerobic, and anaerobic were negative. Pathology demonstrated interstitial fibrosis with intrapulmonary ossifications. Since this entity can be associated with mitral stenosis, a cardiology consultation was obtained and an echocardiogram was performed. This study revealed normal LV size with normal aortic and mitral valves, and contractility. Final diagnosis was interstitial pulmonary fibrosis with intrapulmonary ossifications of the racemic type. The patient has had long term regular follow-up with chest x-rays every six months for four years on continued full duty flying status. The patient was treated with prednisone. Chest x-rays and pulmonary function studies did not show evidence of progression of disease. He has been stable on periodic follow-up on flying status. He presented with shingles on a recent visit without laboratory evidence of immunosuppression. The diagnosis of interstitial fibrosis with over four years follow-up has not to this date prevented flying duty in helicopters. The patient feels healthy, and enjoys a normal life-style with fit for duty status.

DISCUSSION

Interstitial pulmonary disorders can be a chronic process without specific etiology. Past medical history can be helpful or confusing. The patient may have had exposure to infective agents, physical or chemical agents, asbestos, or even immunosuppressive drugs. In this case the patient had recurrent hemoptysis, a diffuse interstitial disorder on chest x-ray, negative lung tissue cultures, asbestos exposure, and long term contact with pet bids including a parrot. Transbronchial biopsy is helpful, but is most often not enough for diagnosis. Routine laboratory studies and pulmonary function studies may not help depending on the stage of disease progression. The interstitial fibrosis disorders are separated into idiopathic pulmonary fibrosis, desquamative interstitial pneumonia, and lymphoid interstitial pneumonia. Open lung biopsy is indicated for diagnosis, and to differentiate idiopathic pulmonary interstitial fibrosis from the other types. The interstitial disorders can be clinically classified as acute for infectious processes, subacute as in sarcoidosis, or chronic as in interstitial fibrosis, or late stage as in honeycomb lung.

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The opinions expressed in this report are those of the autor and do not necessarily reflect the views of the USPHS or the US Coast Guard.

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(1) Commander, USPHS, FS, USCG Flight Surgeon, P.O. Box 2542, Sitka, AK 99835.

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American Society for Circumpolar Health

Sven O. E. Ebbesson, President

First let me thank you for all the support, for all the letters to legislators in Juneau and Washington. Thanks to you, real progress is being made in improving the awareness of the needs in Arctic health research and education.

The purpose of our society is to promote research, education and communication related to Arctic health. My mission has therefore been to reach out and with your help, to change the status quo. I have previously spelled out the need for more health related research and education and the previous lack of interest by the University of Alaska. Major changes are now taking place in the latter, thanks to your letters and the participation by many of you in a number of University initiatives.

The members of our Society clearly believe in doing, not just talking about change. It is therefore that my hat comes off to the volunteers, physicians, nurses and professors in the University initiated Alaska Siberia Medical Research Program (ASMRP). They have volunteered to participate in research projects in Alaskan villages and Siberia ranging from epidemiological studies on diabetes, coronary heart disease, and seasonal affective disorders to physiological, genetic or behavioral studies aimed at understanding the mechanisms underlying ethnic variability in predisposition to disease. Volunteers in these projects have taken weeks, sometimes months, away from family and friends to work under primitive conditions, sleeping on cold gymnasium floors or rickety cots. Always enthusiastic, I've never heard a grumble from a single person in the legions that have participated. The results should be rewarding because major insights in every area have been gained. Although most of the results are to be published this year, a number of papers are already out. A separate manuscript reviewing our findings is being prepared now.

Then there is the generosity of volunteers and institutions. The Fairbanks Memorial Hospital with Jim Gingerich, Theresa Lyons and Donna Woodkey collected medical and research equipment to fill three forty-foot containers that were delivered to the Russian Academy of Medical Science as part of the ASMRP.

The implications of the work of the ASMRP for the future of Arctic Health Research are important, not only because scientists and volunteers from the various campuses of the University of Alaska, the Indian Health Service, the Alaska Department of Health and Social Services, CDC, the Universities of Washington, Virginia, Indiana and Oklahoma, the Medlantic Research Laboratory, and the Veterans Administration joined hands with researchers of the Russian Academy of Medical Science to work on common health programs, but the preliminary studies revealed our ignorance, in fact, provided evidence of our ignorance.

The new insights have made it easier for me to sell the need for more research and education, not only at the University, but to the Arctic Research Commission (ARC) where its chairman, Dr. Donald O'Dowd, has really gone to bat for us. The ARC and Senators Stevens and Murkowski and Representative Young put on a minisymposium for the directors of the National Institutes of Health last year in which five of us Alaskans spelled out the needs and opportunities. Since that time, I've spent a lot of time with NIH directors explaining further the need for funding in the area of Arctic Health Research. The gratifying result is a significant elevation in awareness and enthusiastic support for our various initiatives.

One of these initiatives is the "Partnership Program" between the Dr. James Snow's National Institute of Deafness and Communication Disorders and the University of Alaska. This program, run by Dr. Lawrence Duffy in Alaska, promotes collaboration between their intramural researchers and ours and allows us to send minority students to NIH for training and motivation.

Another initiative that Dr. Harold Varmus, Director of NIH, has approved for funding is a Center Grant for establishing an Arctic Health Research Center at the University of Alaska. I am now finalizing the arrangements for this with his office. Needless to say, this infusion of funds will allow us to fill some of the major gaps in Arctic Health Research. The Center will build on the infrastructure of the ASMRP and add the dimension of education, especially outreach in public education, undergraduate and graduate education.

I have saved the best for the last. The planning for the Xth International Congress of Circumpolar Health, which we are hosting in Anchorage in May, 1996, is progressing very well under the able leadership of Dr. John Middaugh. He informs me that we expect some 750 attendees. We are especially grateful to Dr. Middaugh and his committee for the magnificent work they are

(continued on page 156)

GLIMPSES OF ALASKAN MEDICAL HISTORY

Edited by Robert Fortuine, M.D.

SYPHILIS CONTROL IN RUSSIAN AMERICA - 2 (1844-1846)

Alexander Friedrich Frankenhaeuser (or Frankengeizer, as he is sometimes known) was born in Viborg, in what is now Finland, in 1811. He studied medicine at Dorpat University, in modern-day Estonia, and took his degree in 1836, later taking also the advanced M.D. degree. After a stint as a military physician in St. Petersburg, he signed on with the Russian-American Company and arrived in Sitka in April 1841, where he joined Romanovskii as the second physician at the capital. He too was a collector, like Romanovskii and Blaschke, all of them perhaps inspired by their friendship with the scientist I. G. Voznesenskii, who lived in the colonies from 1839-49. While in Russian America Frankenhaeuser married the daughter of his household manager and the couple had three children born in Sitka and five more after they returned home in 1852. His wife died in 1925, just a few months short of her 100th birthday.

Frankenhaeuser's medical reports, published together with those of Romanovskii, cover the four-year period May 1, 1844, to May 1, 1848, and deal at length with syphilis, tuberculosis, vaccination policy, chronic diseases, and epidemics of respiratory disease (probably influenza), tonsilitis, and blepharitis. Like his colleague, Frankenhaeuser was particularly interested in the syphilis situation and gave further information on the rather draconian control measures that had been instituted in the colonies.

[1844-1845] "Syphilis appeared in Sitkha in spite of all measures, not as frequently as in former years, but nevertheless sufficiently often: the reason for this is the impossibility of using here to their utmost measures which are fully successful in other parts of our North American colonies. Thus, for example, when venereal disease appeared in Kad'iak in 1840 it was . . . quickly eliminated. . . . Equally this disease does not occur at all in the Unalashka and Atkha sections and on the Kurile Islands, because all these points have contact only with Russian vessels, whose crews, on sailing from Sitkha, are examined carefully; and if the disease is discovered there, the colonial management can issue all necessary orders to eliminate it. In Sitkha on the other hand, the Kolosh [Tlingit] . . . are entirely independent from the Russians, and not subject to measures taken by the Management for this purpose.

"In Novo-Arkhangel'sk almost the only victims of syphilis are men who cohabit with the Kolosh women, whereas not one woman in this settlement had syphilis during the year. In order to wipe out the evil at its very root, by order of the Chief Manager, for three successive years the meeting places of the promyshlenniks for debauch with Kolosh women were surrounded with armed detachments, and the women seized were sent to the fort. There, after strict medical examination, the healthy were released, and the infected were held until they were cured. This measure, the only one which can succeed, at first caused strong dissatisfaction among the Toens [elders], who own most of these slave women, and they agreed to their stay in the fort only as long as they were not needed for fish catching or other labors, when they persistently demanded their release, in spite of all warnings. This medical-police measure, the only one available for the time being—taking the women into the fort by force—already shows success. In 1841, the year when this measure began, 62 persons infected with syphilis entered the male hospital, in 1842, 40, and in 1843 only 24 persons, of whom 4 suffered from chronic venereal disease. These latter, constituting only 1/3 of the total sick in 1841, must be considered insignificant, since there are 1,270 inhabitants in Novo-Arkhangel'sk, and most of our promyshlenniks lead a bachelor life and cohabit indiscriminately. In other parts of the colonies, as already noted, syphilis is almost everywhere eliminated, even among the savages near the rivers Kvikhpak [Yukon] and Kuskokhvlek (Kuskokwim?), according to Fleet Lieutenant Zagoskin, after his two-year expedition."

[1845-1846] "Measures for reducing syphilis continued during this year; 17 infected Kolosh women seized at the rendezvous in the forest were treated in the fort. The number of syphilitic men hospitalized during the year increased by five in 1844-1845, probably because many of our workmen were usually in the forest, where they could freely cohabit with the Kolosh women. By the second year not a single infected woman remained in Novo-Arkhangel'sk, and according to word received in the autumn of 1845 from other sections of the Colonies, no venereal disease appeared there."

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History of Medicine in Alaska

THOMAS KIESTER, M.D.

Tom was born on Friday May 13, 1927 in Hamilton, Indiana north of Fort Wayne. His future close friend and associate George vonWichman was also born in 1927 on April 13.

When Tom was twelve his family moved to Springfield, Ohio, where he enjoyed the out-of-doors, fishing, roaming fields and learning to swim in the creek by his house. In June 1995 he attended the 50th reunion of his high school class in Springfield.

Tom Kiester enlisted in the Army on July 7, 1945 and was to serve 17 months and one week, just three weeks shy of enough to avoid the doctor draft later. These three weeks not served would determine the major course of Tom's life.

He was sent to Germany as an enlisted man, where he was in charge of the regimental post office. Tom had one ski vacation in Switzerland and a 10-day furlough which enabled him to see Paris and London.

When he returned to the USA in November 1946, he began to think deeply about a career. He was interested in science. He considered medicine a science. He was to enjoy it but found it frustrating because one rarely knew enough to be satisfied intellectually. Internal medicine was too empirical and often struck him as voodoo because of a lack of objective basis for treatment. In orthopedic surgery it seemed at least possible to evaluate a patient accurately. However, even radiography did not help much when a patient needed an operation for a painful condition. There was always the question whether the optimal treatment had been chosen; for example, fusion of a joint to relieve pain but at the cost of loss of motion. Problem solving is the best part of medicine but frustration as well as satisfaction is always there.

Tom Kiester attended Wittenberg College in Springfield. He had been accepted at Ohio State University, but the large classes produced by the GI Bill of Rights made tiny Wittenberg College more attractive. He lived at home, had a scholarship, worked at many jobs after classes and on weekends, and was graduated in 1950.

Tom went on to the University of Maryland School of Medicine in Baltimore, using the GI Bill for this. Here he met Harriet Warner. They were married after his third year. Dr. Kiester interned at the U.S. Marine Hospital in Baltimore, after which he entered the U.S. Public Health Service because he lacked those three weeks of the required 18 months of military service. He was sent to Sitka.

Harriet and he drove the Alcan Highway to Haines, then took the ferry on to Juneau. From there they flew to Sitka in a Grumman Goose. Fewer than 2,000 people lived there at that time. Fishing was the main source of revenue. There was no logging or tourism. There were, however, small boats, fishing, ducks, deer and exploring. Also there were Drs. George vonWichman and Robert Fraser, who were starting their long Alaska careers by sharing bachelor quarters in the other half of the duplex. The Alaska Steamship Company supplied necessities. A ship arrived every two weeks - always a big day.

Tom recalls a deer hunt with thoracic surgeon Lyman Hunt in the fall of 1957. They encountered a wild storm in a 16-foot skiff. Waves were huge and visibility nil. This was Tom's first experience with big waters - a harbinger of things to come.

Mt. Edgecumbe Hospital at Sitka was the premier medical facility for Alaska Natives, particularly for those with pulmonary and bone and joint tuberculosis. During his first year there Tom assisted at pulmonary surgery. In the second year he was assigned to the outpatient general medical clinic and to the obstetrical service, which he enjoyed.

He spent a memorable Labor Day weekend in 1956 on duty. One patient was a very large 18-year-old primagravida. False labor began on Saturday evening. True labor began Sunday morning, eventuating in a 11 1/2 lb. baby. Back in their duplex, Harriet ruptured her membranes on Labor Day and went into labor. Tom, having lost sleep over his Native patient, was eager for Harriet to get to the hospital to the care of his colleague for delivery. But that doctor's Jeep would not start. At about 6 AM on Tuesday, Tom took Harriet to the hospital in their Jeep. Shortly after arrival, he delivered Paul Thomas. This was truly a labor weekend.

Mt. Edgecumbe was a turning point for Dr. Tom Kiester. Dr. Philip Moore was chief of the orthopedic service. Dr. George vonWichman was a contract physician for the Alaska Territorial Health Department and served under Dr. Moore as an orthopedic surgeon. There were 60 beds for children at the hospital. Tom became fascinated with the orthopedic problems of children even though he had had no experience with orthopedic surgery. Although he was leaning toward internal medicine, he began to be drawn to orthopedics. Even more meaningful was the exposure to mountains and the sea. Tom was hooked on these.

He departed for a four-year orthopedic residency at the University of Louisville Hospital knowing exactly what he wanted to be and where he was going to practice.

Lisa Ann and David Andrew were born in Louisville. And just before the end of his training George put Tom in touch with the Doctors Clinic in Anchorage. He joined the clinic but left after two years because he did not feel comfortable taking general medicine night call, which the clinic insisted upon.

First he had an office of his own in Anchorage on E. Northern Lights Boulevard, but after two years, he joined George vonWichman in an office on W. Northern Lights Boulevard. In 1970 they opened the Anchorage Fracture & Orthopedic Clinic at 36th Avenue and LaTouche. Dr. William Edwards soon joined them, followed by others.

In 1971, George had a Worker's Compensation patient who felt that he was not being treated fairly. He called Dr. vonWichman from the lobby of Providence Hospital. George went to see him between surgeries. The man fired a shot from a revolver into the floor, grazing his own leg. He then marched Dr. vonWichman at gunpoint to the Emergency Room and ordered him to call Governor Bill Egan. When Tom, who had been assisting George at surgery, got wind of this, he went to the ER to help George. He found them seated together at a desk. Tom went in behind the front counter in the ER. He knew the angry patient from having attended him while George was away. From behind the front counter of the ER he called to him by name, "Bob, what is the problem?" The patient was pointing a gun at George. As Bob got up and moved behind the counter away from Tom, George managed to slip out the door.

Tom felt sure that he could use a Judo maneuver to disarm Bob. He asked the patient to let him look at the leg, which was bleeding. At this juncture Bob shouted, "Where's Wichman?" and Sergeant Good of the Anchorage Police Department appeared saying, "Don't move!" The patient turned his pistol toward the officer, who was just behind Tom's right shoulder. The police officer shot twice. Tom was running down the corridor. Bob was dead.

Tom was surprised two weeks later when at general medical staff meeting he was presented a plaque honoring him for his valor. He was doubly surprised inasmuch as he was secretary and the presentation was not on the agenda.

Carol Clouse, Tom's present wife, has an admirable background of her own. She earned an RN in 1969, a degree in accounting in 1980, and a master's degree in teaching celestial navigation in 1988. In 1988 she climbed Denali. She also is a licensed private pilot.

Tom, Carol, three friends, and a cat named Ksenija (Russian for woman) planned a sailing trip from San Diego to Puente del Este, Uruguay by way of Cape Horn. They departed early in November 1990. The woman who was to be cook became seasick and remained so until finally persuaded to take medication on the 23rd day. While this stopped the seasickness, she became depressed and lost 35 lbs. Friction among the crew and a leaking transmission compounded problems. Changing course, they made for Honolulu. By this time no one except Tom wanted to continue.

Tom got the transmission rebuilt, replaced the boat's head, installed an additional cockpit drain, replaced the steering mechanism, and restocked the boat. It was rigged so that one person could handle it.

He cast off on his solo voyage on Sunday, December 16, 1990 at 7 AM. Tom felt well and confident. Carol knew his route and plans. He promised to call whenever possible, but he was never able to get a response from the marine operator in San Francisco before crossing the Equator at 125° West Longitude. It took Tom almost a month to reach the point at 5° North Latitude, 125° West Longitude from which he and his crew had deviated earlier to reach Hawaii in less than two weeks. Equatorial currents and trade winds made windward progress slow.

His first problem was losing a storm trisail in a squall early on. Next, shortly after crossing the Equator, he was thrown from the deck by an unexpected wave and was snapped up short by his lifelines and safety tether to the mast. The most vivid feature of this episode was that Tom instantly sensed, "I was going overboard and was surprised at not being in the water." It happened so fast that he could not even catch shrouds as he was swept by them. Tom lost his cap and his glasses.

But an altogether scarier event was on another day when he had to leave the boat to go beneath it. The boat was becalmed, and the trolling generator line had wound about the rudder and propeller. Tom had no choice. He inflated the portable dinghy and donned scuba gear. There were two lines from Tom to his craft. He was not going to be left behind! He freed the tangled lines and climbed back aboard greatly relieved.

He saw no sharks during the voyage.

Things went along fairly nicely until 135° S, 125° W. At this point he turned toward Cape Horn on a southerly course running with the wind on the starboard quarter. Weather was uniformly overcast, winds 25-35 knots, with long, regular, uniform seaswells from the southwest. Easy sailing lasted until one evening when the boat was struck by a rogue wave, which fell upon the deck portside. It drove the craft downward and hurled it to starboard. Tom was below sitting on a stool at the navigation desk. He did not hear or see the wave coming. "I found myself flung across the boat on top of the galley counter above the refrigerator along with everything else that was loose in the cabin. Luckily I suffered only a broken rib." The boat sustained considerably more injury: the dodger (canvas cover over the cockpit) ripped loose; the traveller mechanism (controls position of the boom) broke; the dinghy mounting broke; the boom bang (which hold the boom to the deck) was ripped loose; several deck-lashed water jugs were gone; and the stove pipe for the galley was torn loose letting water pour through the opening. And because the cockpit hatch was open a few inches, considerable water came in. He lost another sail from a "sausage bag" on deck. The windspeed indicator was blown from the top of the mast. He lost the genoa halyard when the snap shacker broke and the sail fell down. Another problem was that the trolling generator seized, and Tom was unable to start the engine. As a result, by the time he reached the Horn, he had no battery power and was thus deprived of the security of his radio and other instruments.

Before this loss he received the weatherfax from Chile. It had shown a uniform pattern of one low after another passing west to east around the Horn day after day. Weather actually experienced mirrored this perfectly.

Tom did not see Cape Horn during the night of March 10 when he passed it. The next day he identified the Staten Islands and made the decision to head for the Falklands, five days away, rather than to Puente del Este, Uruguay, one week farther. "Too many things were broken, and I was tired."

He gained the Falklands close to the entrance to the Bay of Port Stanley. He recognized it from a video he had seen before departure. It was a welcome sight.

Fog descended, however, before he actually reached the bay. So he sailed on past a narrow headland, entering an adjoining bay. He saw ships anchored and made his way slowly toward them. Before reaching them, he saw lights on shore which he thought were entry lights to the inner harbor. As he sailed past the anchored ships in the dark, he realized that the lights were not navigational lights. He was at the point of turning the boat around when it went softly aground. It stopped without damage because he had been moving very slowly with a light offshore breeze. It was 7 PM, March 15, 89 days after leaving Hawaii more than 8,000 miles away.

He fired off flares because he was almost within hailing distance of the nearest ships. He thought that they would send a tender to tow him free. But there was no response. It turned out that they were squid fishing boats of several nationalities and without tenders.

The tide went out. The boat was on its side by daybreak. There was no wind, no sea. By full daylight, the boat was nearly afloat. A tender from Port Stanley, moving people to and from the anchored ships, notified the harbormaster. He sent immigration officials in a small craft to welcome Dr. Kiester to the Falkland Islands.

A man with an unintelligible British accent was also sent down to the beach. Tom decided not to leave his boat. High tide came and went.

Early next morning, Robin Goodwin returned in the tender to say that he would come at high tide to help. He had asked his boss if he could help Tom on his own time with the Falkland Island Company tender. Since Tom had no power and there was no wind, he was towed to Port Stanley.

Tom was treated nicely by many people, including the Captain of the Port. The Harbormaster, however, claimed that it was the responsibility of the police to rescue him because he was aground. The police declared that he was the harbor's problem inasmuch as he was afloat. This accounted for the long time aground. Furthermore, the Harbormaster chided him for sailing solo.

How would you sum this up? A tale of much courage, which it surely is. A story of a thinking man making appropriate decisions? It is also this. Perhaps you would conclude that it is a tale of extraordinary balance.

Gwynneth Gminder Wilson
Alaska Medical Alliance

(continued from page 132 - Home Care)

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(continued from page 152 - American Society for Circumpolar Health)

doing. I also want to thank the officers and executive board of our society for the excellent work they have done this year. We are also grateful to Dr. Frank Pauls for his work on behalf of the Board.

Let me say again what a pleasure it is to be part of such a vibrant and forward looking Society. I look forward to seeing you all at the Congress in May.

Sven Ebbesson
President

From Out of the Past. . . Over 30 Years Ago

Gloria Park, M.D.

Cover of *Alaska Medicine,* Volume 7 No. 2. University of Alaska-Fairbanks. Scene of the 20th annual ASMA Convention, 1965

ASMA 1965 President
Royce Morgan, M.D.

SCIENTIFIC ARTICLES

"Tuberculosis in Alaska in 1965" by Robert Fraser, M.D.

Dr. Fraser

"Tuberculosis Meningitis" - 11 cases by E. C. Graenshen, M.D."

" Vascular Surgery in Alaska" by Michael Hein, M.D.

"Respiratory Disease Epidemic in Hoonah" by James Justice, M.D.

"The Use of DMSO" by Paul Isaak, M.D.

"Meningococcus Outbreak at Barrow" by Dwayne Reid, M.D.

"The Early Treatment of Chest Injuries" by Arndt Von Hippel, M.D.

Chart No. 1

NEW ACTIVE AND PROBABLY ACTIVE CASES AND CASE RATES, ALASKA AND U.S.
1952 - 1964

Year	Alaska All Races Number	Alaska Native Number	Alaska All Races Rate	Alaska Native Rate	U.S. Rate
1952	743	649	378.5	1854.3	55.0
1953	622	533	293.4	1480.5	53.0
1954	760	651	348.6	1759.4	49.3
1955	661	585	299.1	1539.4	46.9
1956	595	500	270.5	1282.0	41.6
1957	431	369	189.0	922.5	39.2
1958	429	367	120.2	895.1	36.5
1959	356	299	161.8	719.2	32.5
1960	224	188	98.2	413.9	30.8
1961	255	193	108.1	438.6	29.4
1962	323	260	135.5	577.7	28.7
1963	424	336	172.3	730.4	
1964	414	329	165.6	700.0	

Rate per 100,000 population. Alaska rates compiled by TB CONTROL UNIT, ALASKA DEPARTMENT OF HEALTH AND WELFARE, using ADH vital Statistics population estimates for Alaska (All Races) and Tuberculosis Control Unit's for Alaska Native Race.

Paul Isaak, MD

EDITORIAL & REPORTS

A 200 bed Package Disaster Hospital arrived in Ketchikan about June 16, 1965 to be stored in the old Ketchikan Hospital. It is valued at $45,000. Nine other Alaska communities also have packages - Sitka, Juneau, Homer, Kenai, Cordova, Nome, Seward, two in Anchorage and two in Fairbanks.

There was a ground-breaking ceremony September 24th of the Soldotna General Hospital.

Dr. Milo Fritz reported on an EENT clinic held at Sitka on July 1965.

"Children's Bureau Programs in Alaska" by Jack Lesh, M.D.

Authority and responsibility for maternal and child health services in Alaska was assigned to the Department of Health and Welfare by the State Organization Act of 1959.

"Functions and Services of the Public Health Laboratories in Alaska" by Ralph Williams, DPH.

"The Emergency Health Services" by Larry Belmont, Health Mobilization Consultant, Juneau Alaska. Regarding disaster preparation, medical self-help and packaged disaster hospitals.

Arne Beltz, PHN

Amos Alter reported on the Environmental Health Branch of the Alaska Division of Public Health.

Arne Beltz, PHN reported on the Public Health Nurse program in Anchorage.

Mr. Vernon Walker was appointed first ASMA executive secretary.

MUKTUK MORSELS

by Helen Whaley, M.D.

Three psychiatrists from the University of Utah who were enroute to Anchorage in 1964 when the Good Friday Earthquake occurred and were diverted in midair back home, defied superstition and returned March 27, 1965 to hold a seminar.

Drs. James and A.N. Wilson of Ketchikan were taken by helicopter to the Granduc mine disaster near Stewart, B.C. in February to care for a miner who survived burial under the avalanche for 77 hours. Dr. William Wills flew to Ketchikan to help.

Dr. Howard Romig of Anchorage was reappointed to the State Board of Fish and Game.

Dr. Joe Deisher left Seward for the Marshall Islands and Dr. John Fenger of Homer also went to the South Seas. Dr. Herb James arrived in Seward.

Construction began on a new $3,000,000 Arctic Health Research Center at UAF which will include a 40,000 sq. ft. concrete laboratory.

Dr. Clarence Bailey left Palmer for Arizona.

Drs. Bob Johnson of Kodiak and Bob Whaley of Anchorage sailed a ketch from Seattle to Kodiak.

Fairbanks reported that in the past three years 14 MDs have come and gone plus Dr. Tatum left after several years in Fairbanks.

New physicians in Anchorage are Charles Manwiller, Gary Archer, Arndt and Marianne Von Hipple.

Dr. Bob Billings of Anchorage had a major fire in his office and Dr. Royce Morgan escaped from his burning cabin at Big Lake.

Dr. George Wichman spent several weeks near the top of Mt. McKinley but severe storms kept him from reaching the top.

Dr. Gordon Benner of Mount Edgecombe was in a group that climbed the NW ridge of Mount St. Elias.

Consultants from the Mayo Clinic held cardiac clinics in Anchorage, Fairbanks and Sitka. A tuberculosis seminar was held at Alaska Methodist University and an OB-gyn conference was held at Big Lake.

The new children's ward at API was dedicated September 9th.

Dr. Elizabeth Tower became a full time health officer of the Alaska Department of Health and Welfare.

The Navy Legion of Merit was awarded to Dr. William J. Mills, Jr. for exceptionally meritorious conduct in the performance of outstanding contributions in the field of cold weather medicine and surgery. This is the Navy's second highest peacetime award.

After 12 years in Bethel Dr. Harriet Schirmer has moved to upstate New York.

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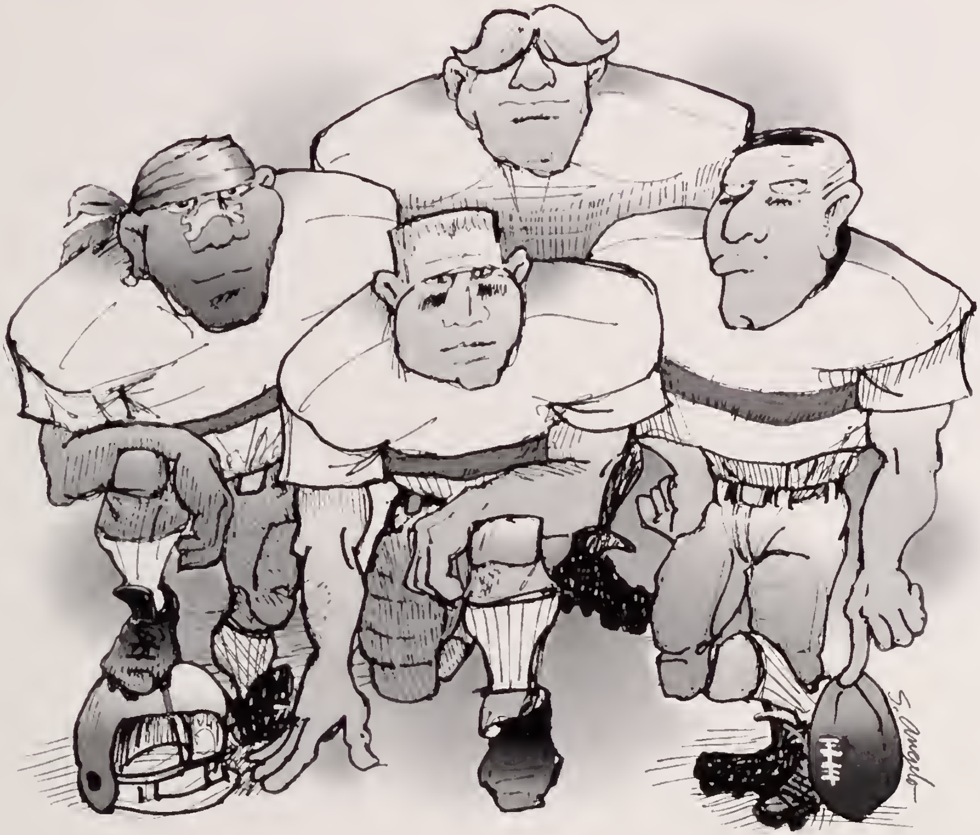

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Volume 38, Number 1

January/February/March 1996

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ALASKA MEDICINE

Official Journal of: Alaska State Medical Association
American Society for Circumpolar Health

Volume 38 **January/February/March 1996** **Number 1**

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About the cover: The Alaska Tobacco Control Alliance bus (part of the Anchorage People Mover system) has been carrying a tobacco-free message since February 1995. The sign on the back of the bus, which currently reads "Tobacco Taxes Save Lives," is changed periodically. The bus was designed by Peter Dunlap-Shohl. Painting was done by Alaska Neon Design. Funding is provided by the Robert Wood Johnson Foundation through a grant to the Alaska Native Health Board. Photos were taken by Chris Arend.

TOBACCO:
Alaska's Most Preventable Health Problem

I wish to thank the Alaska State Medical Association for devoting an entire issue of this journal to tobacco, and for the opportunity to be guest editor. The response of contributors both within and outside of Alaska, the staff at the ASMA office, and others, has been overwhelmingly enthusiastic and prompt.

Tobacco-related death and disease impose an embarrassing black mark on the health record of this century. As the articles in this issue dramatically demonstrate, Alaska's tobacco statistics are among the worst in the nation. This is particularly embarrassing because Alaska has led the nation in other health issues.

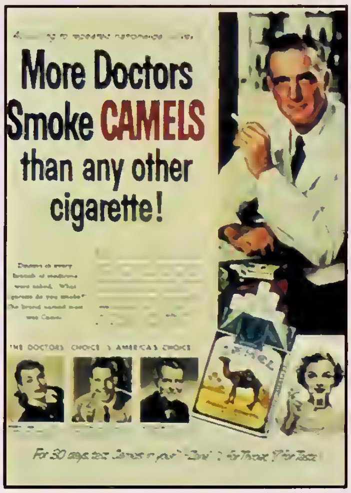
Earlier in this century, medical providers were used to PROMOTE the sale of tobacco. (See accompanying samples of cigarette advertisements*). The industry currently expends nearly $6 BILLION to promote tobacco — a product that when used as directed is guaranteed to kill you. Medical providers have been replaced by Joe Camel and others. Nonetheless, we owe it to our patients, friends, and families to do all we can to eliminate this 20th Century scourge. As deeply ingrained as it is in our society, and with the amount of money leveled at its promotion, this is an ambitious task.

In this issue we have tried to provide as much Alaska-specific data as possible, provide some historical perspective on the problem here, discuss the multi-faceted approaches to tobacco control, and bring comments from tobacco control experts and organizations "outside".

As medical providers having been used in the past to promote the product, let's now "take the lead" and join the Trampling Tobacco Project, the Alaska Tobacco Control Alliance, the Citizens to Protect Kids from Tobacco, the volunteer health organizations, and others, and work toward a Tobacco Free Alaska by the year 2000.

Anne P. Lanier, MD, MPH

*Pictures of tobacco advertisements were graciously provided by D.O.C (Doctors Ought to Care) 5615 Kirby Drive, Suite 440, Houston, TX 77005.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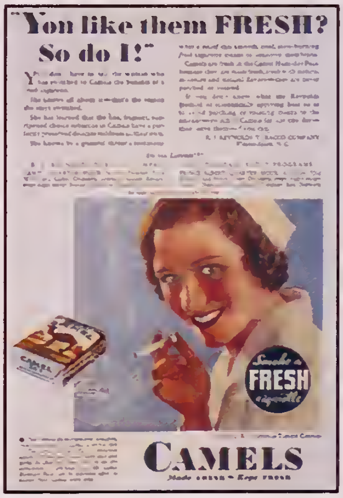

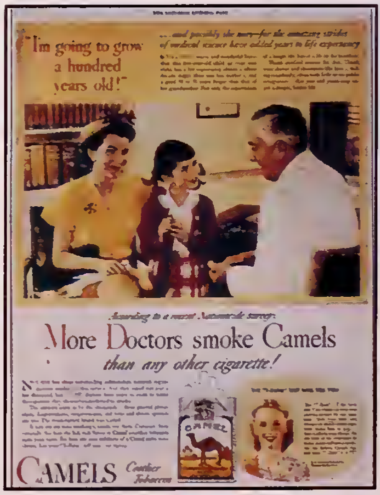
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CAMELS

Editorial

This issue of *Alaska Medicine* is devoted to tobacco related papers. We present historical and cultural perspectives as well as epidemiological data along with statistical data to illustrate that increasing the cost of tobacco results in decreasing the use rate. A recent poll shows that approximately 3/4 of Alaskans support an increase in the current excise tax on tobacco products. This effort should be regarded as a health measure, not a revenue enhancer. It is hoped that no additional revenue will be collected.

In addition to the usual references to the various smoking related cancers and chronic respiratory diseases, my experience as a state medical examiner prompts me to mention deaths due to house fires, and carbon monoxide deaths from smoldering cigarettes.

All members of the state legislature will be furnished a copy of this issue. We hope they will read it and realize that they have an opportunity to make a highly significant contribution to the health of Alaskans for many years to come.

Donald R. Rogers, MD

Funding for the cover photograph and extra pages in this special issue was provided by the Alaska Native Health Board through its SmokeLess States grant from the Robert Wood Johnson Foundation.

Historical Notes on the Introduction of Tobacco into Alaska

Robert Fortuine, MD(1)

THE NORTH PACIFIC REGION

In July 1741, Georg W. Steller, Bering's physician and naturalist, stepped ashore in Alaska, at Kayak Island near the mouth of Prince William Sound. There he left a pound of tobacco and a Chinese pipe at an abandoned Native house site in exchange for a few artifacts he had carried away. A few weeks later, the Russians again offered Chinese pipes and tobacco, this time in person, to some Aleuts who had paddled out to meet their ship in the Shumagin Islands. A sailor even offered one of the Aleuts a lighted pipe, which was said to "displease" him (1). Captain Chirikov, in command of Bering's other vessel, also offered pipes and tobacco to some Aleuts, probably on the island of Adak, but they simply placed the gifts on their baidarkas and paddled away (2).

By 1778, one of Cook's surgeons was able to write that the people of Unalaska "perfectly understood the use of tobacco, which they asked for by that name . . . .(3) Few if any did not smoke, chew, or take snuff. Lieutenant King noted that they preferred tobacco to "all other things;" in fact, some of the Aleut women were willing to bestow sexual favors on members of the crew in exchange for a leaf or two of tobacco (4).

Early Russian fur traders who came to the Aleutians sometimes used tobacco as a gift, a reward, or as an inducement to service, and before long the Aleuts had developed a craving for it (5,6). Since, however, in those early years tobacco was in short supply, the Russians kept it principally for their own enjoyment. The Aleuts seemed to prefer their tobacco in the form of snuff, (7) and there were reports that they would work very hard, often under hazardous conditions, to obtain even a small supply. In the 1820s, the Orthodox missionary Father Veniaminov reported that often more than half of a hunter's income was spent on tobacco, either for chewing or for snuff (8).

Russians also introduced tobacco as an article of trade to the Natives of Kodiak Island perhaps as early as the 1760s (9). Dr. Merck, who visited in the 1780s, found the Koniag eager for snuff, although women less than men (10). By the early 1800s the Kodiak islanders were passionately fond of tobacco and cursing the Russians for having made them so dependent on it. Shortly after 1800 they were using it primarily as snuff placed in the mouth; few sniffed it and no one smoked, according to Davydov (11). Captain Lisianskii around the same time noted that a Koniag would go twenty miles out of his way for a pinch or two of snuff (12). The Russian-American Company also regularly used tobacco as a treat or as an incentive for their Aleut and Koniag hunters stationed at Sitka or at the posts on the Kenai Peninsula. When a new fort was established at Nushagak in 1818, the trader was encouraged to use tobacco among the local Natives to show the good will of the Russians (13).

---

(1) Biomedical Program, University of Alaska Anchorage. This paper is an abridgment and adaptation of Chapter 16 of my book *Chills and Fever: Health and Disease in the Early History of Alaska.* Fairbanks: University of Alaska Press, 1989.

The people of Cook Inlet and Prince William Sound were presumably introduced to tobacco by the English, Russian, and perhaps Spanish explorers and traders who frequented this region in the 1780s and 1790s. The habit, however, did not seem to catch on at first, for both Meares and Billings reported that the Natives showed no interest in the tobacco they offered in trade. (14,15)

In precontact times, the Indians of Southeastern Alaska were known to have cultivated a tobacco-like plant which they mixed with lime and chewed. Lieutenant Whidbey of Vancouver's expedition observed the plant being grown in Chatham Straits, the only crop the Tlingit were known to cultivate. (16) They dried the leaves of this plant over a fire, then ground them up and pressed them into cakes. It was always chewed, rather than smoked or inhaled. After the 1780s, as intensive trade with European ships began, the native plant fell into disuse in favor of the European product, which the Indians found stronger and easier to obtain. They soon learned to smoke it as well as chew it. Tobacco began to take on a ceremonial value and was sometimes used after a shamanistic ceremony, (17) or given to the relatives and friends of a sick man. The Russians handed it out generally as a reward. (18) Tobacco became increasingly popular in the region, and soon leaf tobacco mixed with pulverized bark was being regularly smoked in short, carved wooden or clay pipes. (19)

NORTHERN ALASKA

Curiously, tobacco first reached northern Alaska not by European ships but over traditional trade routes with the Chukchi of Eastern Siberia. According to Ray, the tobacco originated in Circassia, Poland, or Sweden and became an article of trade in the 1750s. (20) Nikolai Daurkin, a Chukchi who visited the Diomedes in the 1760s, reported that the first request of the Natives was for leaf tobacco, for which they were willing to trade expensive furs. (21) In 1789 a large trading market was established at Anyui on the Kolyma River, where the Alaskans traded their furs for tobacco and other European goods, which then passed through several more Native middlemen before reaching the Seward Peninsula. (22)

This trade continued to flourish throughout the nineteenth century, with the major focus on the north coast of Kotzebue Sound and at Port Clarence on the Seward Peninsula. From there tobacco was distributed to the north coast and into the interior via the major river systems. (23)

In the vicinity of Kuskokwim Bay Captain Cook encountered Eskimos in 1778 who were unfamiliar with the use of tobacco. Likewise, when Billings' ship visited Cape Rodney in 1791 the Eskimos were eager to trade for many European goods, but apparently not tobacco. (24) Yet in 1816 the Eskimos of St. Lawrence Island greeted Otto von Kotzebue with a chorus of pleas for tobacco, which they immediately put into their mouths. Later he saw them smoking small stone pipes, about the size of a thimble. (25) The Russians traded freely with the Siberian Yupik of St. Lawrence Island, who were willing to give up an elaborately decorated gut parka for a few leaves, the amount one might consume in a morning. (26) The Eskimos of Shishmaref Inlet also clamored for tobacco, which they seemed to enjoy chewing as well as smoking. This love of tobacco impressed the captain, who found it remarkable that the weed had penetrated where no European vessel had visited. (27)

When a decade later Captain Beechey landed on St. Lawrence Island and at Cape Prince of Wales, he again found tobacco to be the great object of all trade. On one occasion his sailors bought four hundred pounds of caribou meat for four pounds of tobacco. The differing habits of the Natives of the region are illustrated by Beechey's observation that the northern Eskimos generally smoked a short pipe, while some to the south of Bering Strait chewed tobacco, and the St. Lawrence Islanders took it as snuff. The tobacco was often of poor quality and extended with dried wood.(28)

The first European to reach Barrow, also in 1826, found tobacco well known and readily marketable. Eleven years later Thomas Simpson of the Hudson's Bay Company encountered Eskimos to the east of Barrow who immediately clamored for tobacco, of which they were all —men, women, and children—"inordinately fond." At Barrow he found tobacco to be the "grand article in demand." A single inch of it was equivalent in trade to the most valuable articles they possessed.(29)

Robert F. Spencer has presented interesting information on the role of tobacco in traditional Barrow culture. The small pipes they used were of the Siberian or Russian type and were cleaned before use, with a caribou hair placed in the bowl to prevent tobacco flakes from entering the stem. Men's pipes were small and portable, while those of women had longer stems. Bowls might be made of ivory, metal, or clay. Nearly everyone used tobacco in some form, including the children. Besides smoking, many chewed the leaves.(30)

Further south the Eskimos also used tobacco that they had obtained in trade from Siberia and perhaps later from the Russians in the Aleutians, Kodiak, and Prince William Sound. Khromchenko in 1821 found the Bristol Bay and southern Norton Sound Eskimos to be heavy tobacco users,(31) while Glazunov, who in 1833 was the first European to explore the interior of southwestern Alaska, found the Natives already passionately fond of tobacco, which they smoked and took as snuff.(32)

Nelson offers considerable detail on the tobacco habits of the Eskimos of the Seward Peninsula and southward. The women did not often smoke tobacco, but rather chewed it or used it as snuff, whereas the men used all three methods. For chewing, the tobacco was cut into shreds on special boards, mixed with ashes from a tree fungus (obtained from interior Indians), then rolled into pellets, or quids, which they held in their cheek pouch. For smoking, the tobacco was cut very fine. A small tuft of fur was placed at the bottom of the pipe bowl, then a wad of tobacco was stuffed in on top. After lighting the pipe with flint and steel, the smoker took two or three deep draws and held the smoke in the lungs as long as possible. When the oily tobacco extract remaining in the pipestem was cleaned out, it was added to the chewing quids for extra flavor and strength. For snuff the tobacco was finely shredded, thoroughly dried, and then ground into powder in a wooden mortar and pestle. The snuff was then sifted to remove the larger particles. Snuff could either be sniffed or placed inside the lip.(33)

On St. Lawrence Island, near the end of the nineteenth century, Bruce described the Eskimos as "complete slaves to tobacco." All of the men and most women smoked, as well as most children over six. Many of the women also chewed, always swallowing the juice. Old chewing quids were dried and smoked, to extract the last hint of flavor. Snuff was made from finely ground tobacco mixed with pulverized charcoal. He concluded: "An Eskimo who is without tobacco is as wretched as a confirmed drunkard without his whiskey, and he will go to as great extremes to secure it as he would to procure food for himself and family."(34)

Most Athabascan Indians of the interior adopted tobacco directly or indirectly from the Russians. For example, the Han Indians of the upper Yukon were in contact with Russian traders as early as the 1840s, and traveled long distances to obtain tobacco and snuff in exchange for furs.(35) Likewise, the Upper Tanana and the Ahtna groups probably received tobacco in trade from the Russians in Prince William Sound, as well as from the Kluane region,(36) while the Upper Kuskokwim Indians probably obtained it from the Tanaina living around Cook Inlet. The tribes bordering the Eskimos, such as the Ingalik, probably first received tobacco in trade from the Eskimos.(37)

Several of the Athabascan groups also smoked the dried leaves of an indigenous plant, probably even before the arrival of Europeans.(38) The Tanaina chewed a mixture of fungus and cottonwood bark (*Populus balsamifera*), (39) and similar combinations of ashes, dried fungus, or local plants were known from other groups. The Eskimos imported from the Indians the fungus they used for mixing with tobacco.(40)

Athabascan men seemed to be very fond of smoking, at least along the Yukon, and often inhaled deeply,(41) using the small Chinese-type pipe also favored by the Eskimos. Women and children, as well as the men, found delight in chewing.(42)

METHODS OF TOBACCO USE

The Alaska Natives used tobacco in ways that were likely to have been harmful to health. Moreover, the tobacco available to the Natives was crude and the result of primitive methods of curing, and the Natives often adulterated it further with questionable substances such as lime, charcoal, moss, and fungi to "extend" it. Most Natives—men, women, and children—used tobacco whenever they could obtain it, presumably over their lifetime. The tobacco was used and re-used in various ways for economy's sake until the last traces of nicotine and other toxins were extracted.

Murdoch reported that at Barrow nursing children of two or three years were often pacified with a quid of tobacco.(43) Along the Kobuk River mothers sometimes took a child from the breast and put a quid of tobacco or a pipe in its mouth.(44) George Adams had an Athabascan child of five ask him for a chew of tobacco and go off with it as pleased as if it were a piece of candy.(45) Others report Eskimo children of four or five smoking a pipe, taking snuff, or chewing a quid.

The Native manner of smoking must have been particularly harmful. Although several descriptions are available, perhaps the most detailed is that of Capt. C. L. Hooper of the Revenue Cutter *Corwin:*

> "The pipe is lighted with flint, steel and tinder, and the native commences to draw vigorously, swallowing the smoke, which he retains in his lungs as long as possible. A fit of coughing follows, which I at first thought would certainly terminate the life of the smoker in several instances. It is not an unusual occurrence for a native, who has been without tobacco for a long time, to retain the smoke in his lungs until he falls over senseless, having the appearance of a person under the influence of opium. This state lasts but a few minutes, however, when the same performance is gone through with again." (46)

Other early accounts speak of "a momentary stupefaction,"(47) "a stage similar to intoxication,"(48) and "a state of unconsciousness or stupor."(49) Adams described how the Athabascans along the Yukon "give two or three whifs, drawing the smoke down into their lungs, and slowly exhaling it, for a minute after exhaling the smoke, they set like on in a stupor. Their heads drop on their breasts and breathe like one with a severe attack of the Athma [sic]. The young ones when learning to smoke cough for some minutes after smoking very violently. . . ."(50) Seemingly, the stronger the tobacco, the more highly it was regarded. When Glazunov distributed some pipe tobacco and snuff, at the conclusion of a meeting with Indians along the Yukon, "Some were so much dazed by the smoke that they fell unconscious, while others inhaled such a quantity that they could not stop sneezing."(51)

Whatever the hazards of tobacco use, it is clear that the Alaska Natives derived much pleasure from it, and, unfortunately for their health, still do. Adelbert von Chamisso, the German poet and naturalist on the Kotzebue voyage, perhaps described it best:

> "Whoever does not suspect the magic which dwells with [tobacco], let him watch the Eskimo fill his small stone pipe with the precious herb, which he has thriftily mixed half and half with wood shavings, let him see him carefully light it, then eagerly with closed eyes and long, deep puffs breathe the smoke into his lungs and blow it out again into the air. Meanwhile the eyes of all are fixed on him and the one next to him is already stretching out his hand to receive the instrument so that he too may draw a puff of happiness. . . ." (52)

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Historic Ceremonial and Medicinal Use of Tobacco Among American Indians

Donald H. Reece(1)

Tobacco was often used by shamans and in agricultural rites. It was used in the harvesting of crops to bless the harvest. Such a ceremony linked tobacco and the fertility of the land, a strong psychological bond made still stronger by tobacco-induced altered states of consciousness and supernatural visions. The rising smoke of tobacco was regarded as a method by which communication with the gods/creator might be facilitated. The Iroquois believed that smoke carried their petitions to the Great Spirit. The Delaware sacrificed tobacco to ensure success in the hunt. The Crow worshipped the sun, the moon, and tobacco. It was the only thing cultivated to "ensure the continued welfare of the people." Among the Ojibwa/Chippewa, tobacco was placed on a rock to alert the spirit to ward off storms. As stereotypes suggest, tobacco was commonly used to bind agreements between tribes, and it often accompanied invitations to individuals or families. Tobacco was also given as payment to a shaman, obligating him to fulfill the requests of his client.

There was a great deal of ceremony and spirituality involved in the traditional use of tobacco. "In Pawnee ceremonies the pipe was always tamped with an arrow captured from the enemy. It was forbidden to pack it with the fingers, as the gods might think that the man who did so offered himself with the tobacco and take his life." This example illustrates what power tobacco was believed to have had and its overall importance.

In addition to its ceremonial uses, tobacco was traditionally employed medicinally by many different tribes. Accounts from tribes from different areas of the country describe tobacco being used in very similar, if not identical, ceremonies. It served as an analgesic and a treatment for earaches. It was chewed as a remedy for toothaches. Open wounds and the bites of insects or snakes were treated with tobacco because of its presumed antiseptic properties. The Winnebago and the Seminole, along with other tribes, scattered tobacco while repeating prayers to exorcise spirits or ward off the evil influences that caused disease. One Native practice was to blow tobacco smoke into the ear to kill the "Woodland insect" that was believed to cause insanity by drying up the brain. Tobacco was also heralded as a remedy by some for asthma, rheumatism, chills, fevers, intestinal disorders, child birth pains, and headaches.

Study of the calumet, an elaborately decorated clay shaft to which a pipe bowl might be attached, seems to be one of the better ways to assess traditional tobacco use. It was different from other pipes in that the calumet was the source of great ceremonialism and was held to be sacred. The calumet was said to have had the potential to make friends out of mortal enemies and to have provided for peaceful interactions between strangers.

Sources from early periods support the idea that pre-European contact smoking was minimal among American Indians and that they smoked only in moderation for medicinal or spiritual reasons. Pawnee use of tobacco prior to European contact was strictly sacred and ceremonial. The same is said of the Zuni.

An examination of the early smoking practices of some tribes such as the Northern Paiute of the Great Basin reveals than the early Native peoples understood tobacco much better that did the Europeans and later Americans, even after a few hundred years' use. Smoking was practiced only by men; "young boys would not smoke because they were afraid it would impede their ability to pursue game." This suggests that, although tobacco did have spiritual importance in their lives, its powerful physical effects as well as relative scarcity proscribed its use for individual pleasure.

Tobacco continues to play a requisite role in at least two contemporary religious healing ceremonies with deep roots in the past: (1) The Peyote religion (or Native American Church) and (2) the Plains Sun Dance.

The opinions expressed in this paper are those of the author and do not necessarily reflect the views of the Indian Health Service.

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(1) Tobacco Control Coordinator, Indian Health Service, Cancer Prevention & Control Program, Headquarters West, 5300 Homestead Road, Albuquerque, NM 87110

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Anchorage Clears the Air

How Anchorage Came to Ban Smoking in All Buildings Operated by City Government Including Public Schools

Rodman Wilson, MD(1)

January 1, 1987. That was the great day when the Municipality of Anchorage, Alaska, moved boldly to protect the public and its 3,910 city employees by outlawing smoking in all 123 municipally operated buildings. It even decommissioned all smoking lounges. The ordinance establishing the new workplace rule applied equally to 75 public schools and their grounds beginning July 1, 1987.

How did this come to pass? What led the city of Anchorage to do this?

The story began in 1983 when the health and social services departments, housed together in a separate building, decided to eliminate smoking progressively over a nine-month period. These departments wanted to protect the many children, young women and others who came to the building each day for services, wanted to make the workplace safer for employees, and desired finally to be an example to others in the community. The plan did not reach its initial goal of total abolition of smoking but did succeed in confining it to one room in the basement of the five-storied health department building.

Impelled independently, but noting the health department's action, the Alaska Native Medical Center banned smoking altogether in its hospital in Anchorage on January 1, 1986. This was the first hosptial in Alaska to do so. Soon afterwards, smoking was barred in almost all other Indian Health Service facilities and other hospitals in the nation.

Prior to this, in 1984, a state statute concerning smoking in public places became law in Alaska. Among other features it allows "a person in charge" of a building to prohibit smoking or, alternatively, to designate certain portions as smoking or no-smoking, making, however, "reasonable accommodations for the needs of the smokers and non-smokers."

The Municipality of Anchorage found this law unworkable. There was no way to subdivide buildings salubriously. Smoke refused to be gerrymandered. Obeying natural rather than man's law, it diffused freely under doors, over dividers, and through so-called smoke-eaters to occupy, like a drop of ink in a bowl of water, whatever space is available. It also circulated and recirculated to all floors through heat-ventilation-air conditioning (HVAC) systems.

Accordingly, after listening to a presentation in September, 1986 by Robert Rosner, brought to Anchorage by the American Lung Association of Alaska from the Smoking Policy Institute of Seattle Uninversity, Mayor Tony Knowles decided to submit to the Anchorage Assembly an ordinance to prohibit smoking altogether in city operated buildings and schools. The ordinance was endorsed unanimously by the Anchorage School District Board on November 24 and was approved by the Assemby by a vote of 8-3 on December 9 after intense public debate and two spirited public hearings.

Most persuasive of many arguments proffered were those relating to health and public safety. School Board and Assembly members came to realize that breathing someone else's smoke is, indeed, harmful. One week after passage of the ordinance, United States Surgeon General Koop released his forceful report, entitled, "The Health Consequences of Involuntary Smoking." This comprehensive document incriminated sidestream smoke as causing or accelerating numerous childhood and adult disorders including cancer.

Also convincing was the economic argument that it costs an employer several hundred to several thousand dollars more annually to have a smoker on the job than an employee who does not smoke. Costs are in extra time off sick, more permanent disability, early retirement, early death, extra costs for cleaning, shorter life of floor coverings, furniture and precision instruments, increased HVAC costs, and higher premiums for fire, life, and health insurance. Finally, it was realized that morale would improve once divisive wrangling about smoking in the workplace ceased.

How did the new ordinance work? Surprisingly well. Enforcement was not a problem. Obviously, non-smokers were pleased. They constituted at the time 77% of city employees. Among the 23% who smoked, some forsook cigarettes in the fall in anticipation of the new rule. Others made New Year's resolutions to quit. Still others enrolled in one of several smoking-cessation courses offered in town. Spouses signed up too. By prearrangement, tuition was partially underwritten as a benefit of the municipal health insurance plan. Actual numbers of these several cohorts were not amassed. Others, incorrigibly addicted to tobacco, continued to smoke, though not on "company time" except when in the field. And during their breaks, they huddled outside entry doors or around the corner, as they still do, to smoke. Finally, a few workers said that they would quit their jobs, but probably not many did. Jobs were scarce in Anchorage that winter.

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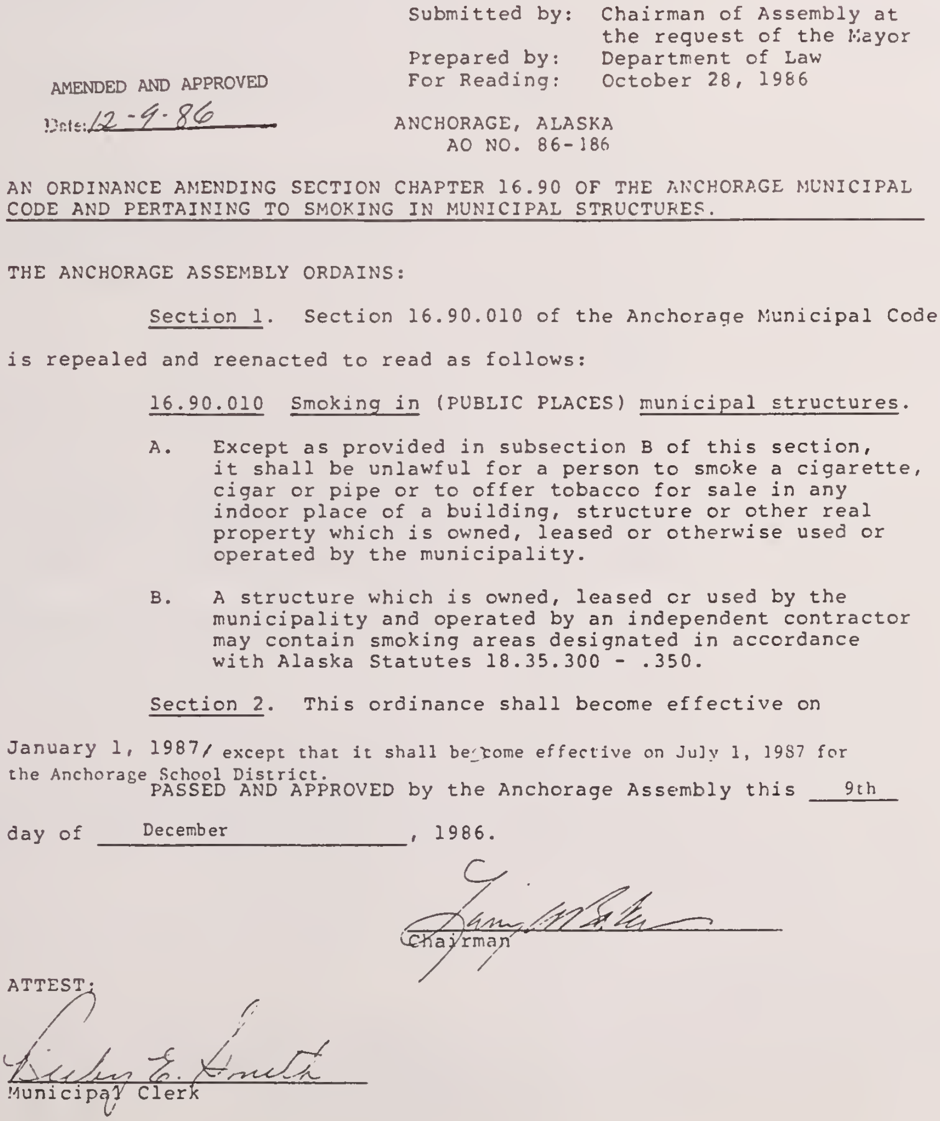
(1) Dr. Wilson was public health director for the Municipality of Anchorage in the mid-1980s. Currently he is acting executive director of ASMA.

Shortly after passage of the ordinance, several businesses and other governmental units followed the city's then radical approach to workplace safety by banning smoking. Among them were several banks, an oil company, a long-distance telephone company, many stores, and the Alaska Court System.

One of the unions representing city employees challenged the new law on the ground that it violated agreed upon conditions of work. The union was represented at arbitration by a prominent Anchorage attorney and brought as its star witness a "scientist" under contract to the Tobacco Institute. His every argument against the ordinance was vigorously countered by the city. The city won. Health and safety superceded convenience of workers.

Dr John Middaugh, State of Alaska Epidemiologist, declared that the move by the Municipality of Anchorage was the greatest step on behalf of public health in Alaska since 1977, when eight percent of school children were turned away from school because their immunizations were not up-to-date. Enforcing compliance with that, then new, requirement put Alaska at the forefront nationally in suppression of childhood infectious diseases. The city's sapient move against smoking, like that of the Alaska Native Medical Center, showed public and private entities across the country the way to heightened workplace safety.



Submitted by: Chairman of Assembly at the request of the Mayor
Prepared by: Department of Law
For Reading: October 28, 1986

AMENDED AND APPROVED
Date: 12-9-86

ANCHORAGE, ALASKA
AO NO. 86-186

AN ORDINANCE AMENDING SECTION CHAPTER 16.90 OF THE ANCHORAGE MUNICIPAL CODE AND PERTAINING TO SMOKING IN MUNICIPAL STRUCTURES.

THE ANCHORAGE ASSEMBLY ORDAINS:

Section 1. Section 16.90.010 of the Anchorage Municipal Code is repealed and reenacted to read as follows:

16.90.010 Smoking in (PUBLIC PLACES) municipal structures.

A. Except as provided in subsection B of this section, it shall be unlawful for a person to smoke a cigarette, cigar or pipe or to offer tobacco for sale in any indoor place of a building, structure or other real property which is owned, leased or otherwise used or operated by the municipality.

B. A structure which is owned, leased or used by the municipality and operated by an independent contractor may contain smoking areas designated in accordance with Alaska Statutes 18.35.300 - .350.

Section 2. This ordinance shall become effective on January 1, 1987, except that it shall become effective on July 1, 1987 for the Anchorage School District.

PASSED AND APPROVED by the Anchorage Assembly this 9th day of December, 1986.

Chairman

ATTEST:

Municipal Clerk

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Smoking Attributable Mortality and Economic Costs in Alaska 1992-94

Catherine Schumacher, MD, MSPH(1)

ABSTRACT

Tobacco is one of the leading preventable cause of death in the United States and Alaska. Alaska has one of the highest smoking prevalences. The Smoking-Attributable Mortality, Morbidity and Economic Cost software developed by the Centers for Disease Control and Prevention was used to estimate the deaths and economic impact due to smoking in Alaska,.

In Alaska during the three year period 1992-94, 1416 deaths were estimated to be attributable to smoking, accounting for 19.8% of the 7159 deaths during that time. Direct medical care costs in 1993 due to smoking related illnesses were estimated at $96.5 million. Additional smoking related costs include indirect mortality costs of $183.2 million and indirect morbidity costs of $15.9 million. The total economic cost attributable to smoking related illness for 1993 is estimated to be $295.6 million. In summary, for the time period 1992-94, smoking was estimated to result in 470 deaths per year and in economic costs of almost $300 million per year.

INTRODUCTION

Tobacco is one of the leading preventable cause of death in the United States (1). The U.S. Centers for Disease Control and Prevention (CDC) estimate that smoking kills approximately 419,000 people in the U.S. each year (2). Deaths that are related to cigarette smoking include a portion of: cardiovascular disease; cancers of the lung, larynx, oral cavity, esophagus, pancreas, bladder, kidney and cervix; chronic bronchitis, emphysema, and other respiratory deaths (2,3). Smoking also results in deaths in the perinatal period because maternal smoking causes a portion of low birth weight infants and preterm deliveries, and has been associated with SIDS.

Alaska has one of the highest smoking prevalence rates in the United States. Alaska's smoking rates (28.1% among men and 25.0% among women) are similar to those found in Nevada and in the tobacco-growing states (4,5). Alaska Natives (46.5% among men and 39.3% among women) have even higher smoking rates (6).

To estimate the deaths and economic impact of smoking in Alaska, we used the Smoking-Attributable Mortality, Morbidity and Economic Cost (SAMMEC) software developed by CDC (3). SAMMEC uses attributable risk formulas to estimate the deaths from neoplastic, cardiovascular, respiratory, and pediatric deaths associated with cigarette smoking. SAMMEC software uses relative risks for current and former smokers for each of the causes of death shown in Table 1.

METHODS

Mortality

SAMMEC was used to estimate the deaths attributable to smoking for Alaska adults (age ≥ 35 years) and infants (age < 1 year) using the 1992-94 mortality data for Alaska. Age-specific smoking prevalences for all races were obtained from the 1994 Behavioral Risk Factor Surveillance Survey (7). Alaska Native prevalence rates were obtained from the 1991-93 combined data from the Alaska Behavioral Risk Factor Surveillance Survey (6) (Table 2).

Economic Costs

Direct health care costs are the costs for prevention and treatment of smoking related diseases. CDC has been using a new method to calculate direct medical care costs which has not yet been included in the SAMMEC software (8). CDC used the method to estimate that in 1990, $76 million was spent in Alaska on smoking related illnesses, which represented 6.13% of Alaska's total health care costs for that year (9,10). In order to estimate the 1993 direct health care costs for Alaska, that percentage was applied to the total medical care costs estimated for 1993. Nationally, about 7.1% of health care costs are attributable to smoking (11).

The SAMMEC program was used to calculate the indirect mortality costs, which are the foregone wages and salaries for persons who die prematurely from smoking related causes for 1993. The national 1990 expected lifetime earnings and housekeeping services by age and sex were used. Indirect morbidity costs were also estimated using SAMMEC, which are the lost earnings and productivity for persons disabled by smoking related illnesses.

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(1) Alaska Division of Public Health, Section of Epidemiology, PO Box 240249, Anchorage, Alaska 99524.

Table 1

Relative Risks* for Death Attributable to Smoking for Current and Former Smokers, by Disease Category and Sex Used by SAMMEC Software Program

Disease Category (ICD-9 code)**	Male Relative Risk		Female Relative Risk	
	Current Smokers	Former Smokers	Current Smokers	Former Smokers
Adult Diseases (≥ age 35)				
Neoplasms				
Lip, oral cavity, pharynx (140-149)	27.5	8.8	5.6	2.9
Esophagus (150)	7.6	5.8	10.3	3.2
Pancreas (157)	2.1	1.1	2.3	1.8
Larynx (161)	10.5	5.2	17.8	11.9
Trachea, lung, bronchus (162)	22.4	9.4	11.9	4.7
Cervix uteri (180)	--	--	2.1	1.9
Urinary Bladder (188)	2.9	1.9	2.6	1.9
Kidney, other urinary (189)	3.0	2.0	1.4	1.2
Cardiovascular Diseases				
Hypertension (401-404)	1.9	1.3	1.7	1.2
Ischemic heart disease (410-414)				
Age 35-64:	2.8	1.8	3.0	1.4
Age 65+:	1.6	1.3	1.6	1.3
Other heart disease (390-398, 415-417, 420-429)	1.9	1.3	1.7	1.2
Cerebrovascular Diseases (430-438)				
Age 35-64:	3.7	1.4	4.8	1.4
Age 65+:	1.9	1.3	1.5	1.0
Atherosclerosis (440)	4.1	2.3	3.0	1.3
Aortic aneurysm (441)	4.1	2.3	3.0	1.3
Other arterial disease (442-448)	4.1	2.3	3.0	1.3
Respiratory diseases				
Pneumonia and influenza (480-487)	2.0	1.6	2.2	1.4
Bronchitis and emphysema (491-492)	9.7	8.8	10.5	7.0
Chronic airway obstruction (496)	9.7	8.8	10.5	7.0
Other respiratory diseases (010-012, 493)	2.0	1.6	2.2	1.4
Pediatric Diseases (age < 1 year)				
Short gestation, low birthweight (765)	1.8***			
Respiratory distress syndrome (769)	1.8			
Other respiratory conditions (770)	1.8			
Sudden infant death syndrome (798)	1.5			
Burn deaths	50% of total burn deaths			

* Relative to never smokers.

** International Classification of Diseases, Ninth Revision.

***Relative risk for infants born to a smoking mother versus infants born to a non-smoking mother.

Table 2.

Smoking Prevalences Used in SAMMEC Calculations

	Gender	Age (Years)	Percent Current Smokers	Percent Former Smokers
All Races*	**Men**	35-64	26.7	36.5
		65+	25.6	49.5
	Women	35-64	30.0	30.1
		65+	17.4	36.2
		Child-bearing ages 18-44***	25.0	
Alaska Native**	**Men**	35-64	44.7	34.8
		65+	38.4	36.7
	Women	35-64	39.2	24.8
		65+	13.2	21.1
		Child-bearing ages 18-44***	44.2	

* Alaska Behavioral Risk Factor Surveillance System, 1994

** Alaska Behavioral Risk Factor Surveillance System, 1991-1993

*** Smoking prevalences used to estimate infant deaths due to maternal smoking

Table 3.

Total Number of Deaths and Smoking Related Deaths by Gender and Cause
Alaska Residents: 1992-94

	Male		Female		Total	
Cause of Death (ICD-9)*	**Number (%)**	**Total**	**Number (%)**	**Total**	**Number (%)**	**Total**
Cardiovascular (390-448)	374 (30.8%)	1214	159 (20.0%)	796	533 (26.5%)	2010
Cancer (140-208)	358 (38.3%)	933	188 (26.0%)	722	546 (33.0%)	1655
Respiratory (460-519)	138 (52.8%)	261	121 (50.0%)	242	260 (51.7%)	503
Perinatal (740-79, 798.0)	8 (7.8%)	103	6 (6.0%)	101	14 (6.9%)	204
Total (All Causes)	920 (21.0%)	4376	496 (17.8%)	2780	1416 (19.8%)	7159

*International Classification of Diseases, Ninth Revision.

RESULTS

Mortality

An estimated 1416 deaths during the three year period 1992-1994 were attributable to smoking, accounting for 19.8% of the 7159 deaths during that time (Table 3). Smoking accounted for 26.5% of all cardiovascular disease deaths, 33.0% of all cancer deaths, 51.7% of all respiratory deaths, and 6.9% of deaths from perinatal causes.

Of the 1402 deaths attributable to smoking among adults, 912 were men, and 490 were women (Table 3). Among women, smoking accounted for a smaller percentage of total deaths caused by cardiovascular disease and by cancer than did smoking among men. The percentage of respiratory deaths were similar in men and women. Alaska Natives account for 23.2% (329) of the smoking related deaths, although they account for 16.5% of the state's population (12). The distribution of smoking related deaths for Alaska Natives is similar to that seen for all Alaskans (Table 4).

Economic Costs

Direct medical care costs in 1993 due to smoking related illnesses were estimated to be $96.49 million (Table 5). Additional smoking related costs were an indirect mortality cost of $183.2 million and indirect morbidity cost of $15.94 million. The total economic cost attributable to smoking related illness for 1993 is estimated to be $295.63 million.

Table 4.
Total Number of Deaths and Smoking Related Deaths in Alaska
Alaska Natives: 1992-94

Cause of Death (ICD-9)*	Number (%)	Total
Cardiovascular (390-448)	105 (27.0%)	388
Cancer (140-208)	115 (35.3%)	326
Respiratory (460-519)	68 (43.6%)	156
Perinatal (790-779,798.0)	7 (10.9%)	64
Total (All causes)	329 (18.0%)	1829

*International Classification of Diseases, Ninth Revision.

DISCUSSION

During 1992-94, there were an average of 470 smoking related deaths each year in Alaska which resulted in economic costs approaching $300 million. Smoking results in more deaths than AIDS, alcohol, aircraft crashes, falls, fires, firearms and motor vehicle crashes (Figure 1).

In the U.S. in general, men have been smoking longer than women and most smoking deaths are caused by long term use of cigarettes. Because smoking rates are now similar for men and women, women may have higher smoking attributable mortality in the future. Alaska Natives are at even higher risk because of their higher smoking rates.

The majority of Alaska smokers (83.7%) began smoking between 10 and 20 years of age (13). In the U.S., by the 1980's, almost no regular smoking began after the age of 18 (14). Therefore, efforts to decrease tobacco use in the U.S. are being directed towards school-age children and adolescents , including limiting advertising and access to cigarettes (15). Additional tobacco control efforts include developing and enacting strong policies for clean indoor air, increasing excise taxes, and increasing educational efforts.

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Table 5.
Economic Costs of Smoking in Alaska: Estimates for 1993

Smoking related direct costs*	$96,490,000
Smoking related indirect mortality costs**	$183,200,000
Smoking related indirect morbidity costs**	$15,940,000
Total smoking related costs	$295,630,000

* Calculation of direct costs based on 6.13% of total medical care costs for Alaska for 1993 ($1,573,000,000)

** Indirect mortality costs calculated using SAMMEC with a 3% discount rate and 1990 earnings data

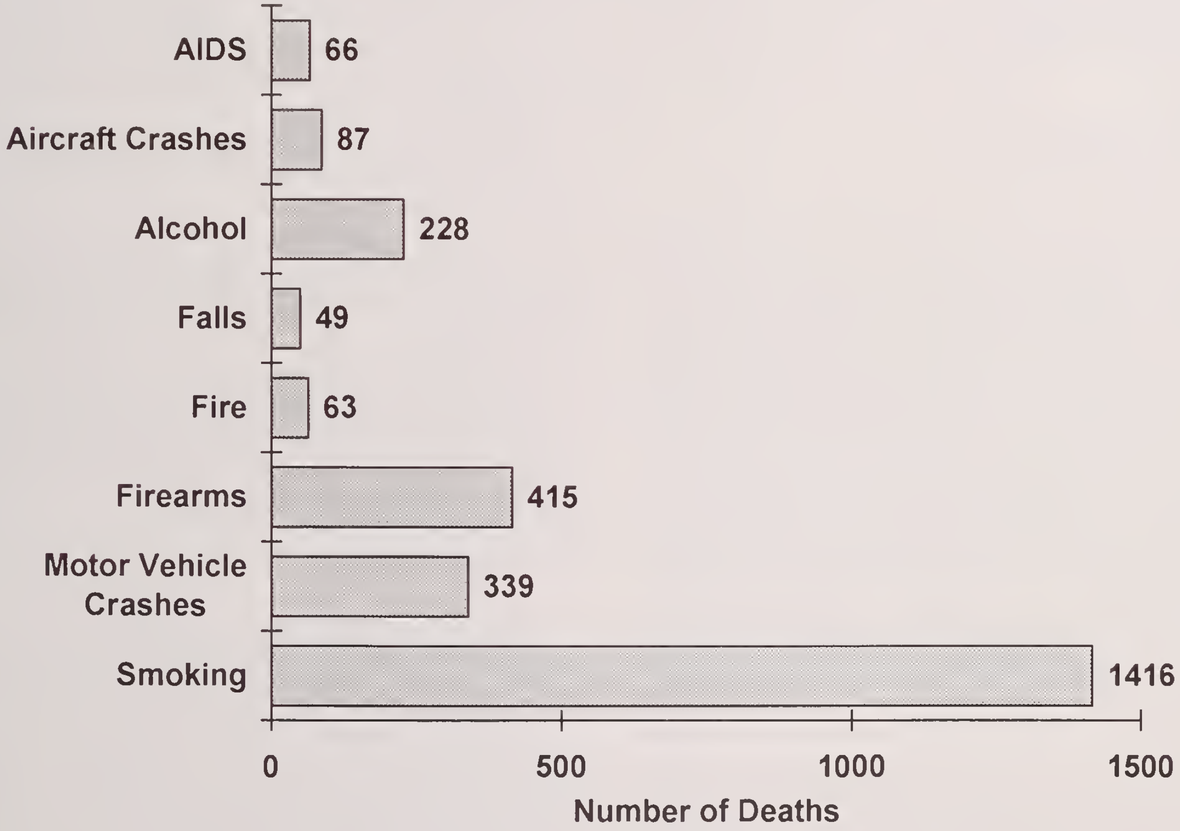

Figure 1: Alaska Resident Deaths by Selected Cause : 1992-94. Mortality Data for Alaska Residents 1992-94, provided by the Bureau of Vital Records, Alaska Division of Public Health. ICD-9 Codes: Aircraft crashes: 840-845; AIDS 42-44 ; Alcohol 291, 303, 305, 357.5, 535.3, 425.5, 790.3, 860, 571.0-571.3; Falls 880-888; Fires 890-899; Firearms 922,955,965,970,985; Motor vehicle crashes 810-825.

Prevention That Works

Peter Nakamura, MD(1)

ABSTRACT

The solution to the problem of tobacco use is complicated when adolescents are faced with numerous risk factors. Alaska has a partnership between federal, state, and private interest groups and individuals who are committed to addressing the problem. This partnership is being expanded through the creation of additional local alliances. Each of the partners has influenced state policies on the control of the use of tobacco products. Because of the difficulty in changing adolescent behavior through education and information there is a need to decrease access to the offending products. A proven way to decrease access and adolescent use of tobacco products is raising the cost of the products through higher excise taxes.

THE PROBLEM

Simply stated, the problem is the increased economic burden to the state and to individuals, decreased quality of life, increased morbidity and early demise associated with tobacco use. It is difficult to imagine anyone who is not at least aware of the basic problem.

Unfortunately, we have a major hiatus between knowledge and practice. I recently had the good fortune to hear Judge Dennis A. Challeen (1) lecture on how people of good sense end up in court. The good judge starts with the explanation that you and most readers of this article are NORPs. A NORP is a normal ordinary responsible person. We take information and process it in a way that leads to the correct choice. Unfortunately, most NORPs undergo NORP WARPS. These are best described as out of character behavior followed by self-correction and return to responsibility. It is during these WARPS that NORPS commit acts that can get them in trouble with the law or other standards set by society. Think of the loyal and faithful husband who was caught in a compromising position the one time he wandered beyond the bounds of holy matrimony. He can be paying for that action for the rest of his life.

This gets us to the underlying cause of THE PROBLEM. Teens are JUNIOR NORPs experiencing a MEGA NORP WARP. This is the time in which they are most vulnerable to high risk experimentation. Nearly 84% of Alaskan adults who smoke started smoking between the ages of 10 and 20. In Alaska, 27% of 12th grade girls and 18% of 12th grade boys reported daily use of cigarettes in a 1989 survey (2).

A SOLUTION

Just imagine if we had no automobiles and roads. The consequence would be that we would have no auto accidents and no traffic violations. The same can be said for tobacco and problems related to its use. However, automobiles, roads, and tobacco will be with us until we come up with a better means of daily transportation and a substance that is equally addictive and as well financed as tobacco.

Tobacco industry advertising has been extremely successful in affecting teenage use of tobacco. (3) Education efforts have not been equally efficient in reversing the trend. Limiting access to tobacco, alcohol, unhealthy practices and high risk behaviors are effective ways to assist our JUNIOR NORPs through the MEGA NORP WARP.

An analysis of countries around the world shows the powerful relationship between price and consumption. (4) The Canadian experience has demonstrated a direct negative correlation between increased tobacco tax and teenage use of cigarettes (Figure 1).

FEDERAL POLICY

The Food and Drug Administration (FDA) Commissioner David Kessler has called smoking addiction among young people " a pediatric disease" and an "epidemic." There has been no progress in reducing teenage smoking rates in the last decade despite continuing progress against adult smoking. The most recent data indicate that smoking among young people actually increased since 1991, with the largest increase among the youngest smokers. There was a 30% increase in smoking among 8th graders between 1991 and 1994. Between 1970 and 1986, the use of snuff increased 15 times and the use of chewing tobacco increased four times among male adolescents ages 17-19. (5)

On August 10, 1995, President Clinton announced proposed rules that will allow the FDA to affect the sale

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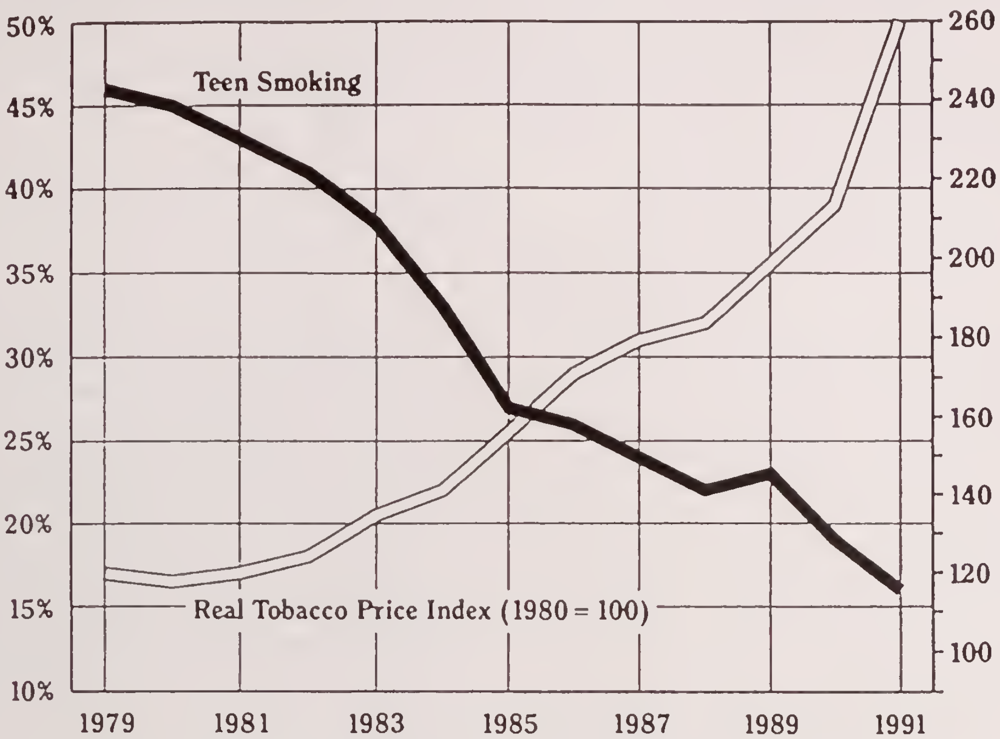
(1) Director, Division of Public Health, State of Alaska, Dept. of Health& Social Services, Juneau, AK 99801.

Figure 1: Real cigarette prices and cigarette smoking among Canadians age 15 to 19



Source: Statistics Canada, Labour Force Survey, 1991; Canadians and Smoking: An Update. *Health and Welfare, Canada, 1991.*

and distribution of nicotine-containing cigarettes and smokeless tobacco product to children and adolescents

The regulations would not only reduce children and adolescent access but will also affect the amount of positive imagery that makes these products so appealing to them. Cigarette vending machines, free samples, mail-order sales, and self-service displays would be prohibited. The objective of the proposed rule is to help the nation meet "Healthy People 2000" objectives on the adolescent use of tobacco products.

Federal Law (the Synar amendment) mandates state level enforcement of laws reducing youth access to tobacco products and requires states to reduce adolescent access to tobacco products. Failure to do so will result in the withdrawal of federal alcohol and substance abuse grant funds. (6) Because of the huge tobacco industry war chest, Federal policies leading to the funding of local tobacco control activities has been critical. CDC and other federal agencies have supported local capacity for tobacco prevention and control activities. CDC has funded 5-year grants to states for Initiatives to Mobilize for the Prevention and Control of Tobacco Use (IMPACT). The Federal Environmental Protection Agency (EPA) provided special funding to the Alaska Health Fairs for health displays, to the Cancer Society for staffing of Smokeless Class 2000.

Not everyone agrees with the role of government in setting health policy, but no one can deny that many of the positive changes experienced could not have taken place without such action. It is, however, critical that the public influence the directions taken by government policy. About fifteen years ago in my efforts to initiate a smoke-free environment in the Portland Federal Building, I found the greatest obstacles to be federal regulations, policies, and personal bias. Thanks to the Clean Indoor Air Act and related regulations, we no longer have to endure irritating and harmful smoke in our work environment, can travel in smoke-free planes, and enjoy a meal without the stench or acrimonious pall of tobacco smoke.

STATE POLICY

Governor Knowles has committed this administration to reducing adolescent use of tobacco. Based on the awareness that high cost of tobacco products will reduce the use of tobacco products by children, he has endorsed an increase in the state tobacco tax. Governor Knowles has stated his hopes that the higher tax "doesn't raise a single dollar" because it is successful in reducing tobacco use.

The role of Alaska's Department of Health and

Social Services in the prevention of tobacco use is within statutory obligations for promoting and preserving public health. The state has a responsibility for compiling data and for producing appropriate information to guide the formulation of sound policy. The state should also provide technical support and access to resources needed for constructive statewide and community based interventions.

The Division of Public Health Behavioral Risk Factor Surveillance System (BRFSS) (7) supported by state and federal funds has for several years collected and analyzed risk behavior information. Information from this and the Youth Behavior Risk Survey recently conducted through the joint effort of the Department of Education and Division of Public Health are reported in this issue. Information gained through these sources as well as other data bases such as the Bureau of Vital Statistics, the Alaska Native Cancer Registry maintained for many years by Dr. Anne Lanier, and the Report of Cancers in Alaska Natives (8) will be available to assist in developing sound tobacco policy for our state.

State tobacco control policy is guided by the Alaska Cancer Control Plan (9) and Healthy Alaskans 2000 (10). The focus of the policy is on reducing youth access to tobacco products, limiting tobacco product advertising, clean air, early childhood education, and the support of tobacco control advocacy activities.

The primary state role in tobacco control activities has been one of facilitation and support and often to get out of the way of the many organizations and individuals commited to the elimination of tobacco use. The Division of Public Health has been successful in competing for federal funds which have in turn been used to support community prevention activities and a statewide tobacco control coalition of over 220 organizational and individual members. The Alaska Tobacco Control Alliance (ATCA) advises the Division of Public Health on strategies, goals, and activities important to the reduction of tobacco use. An additional function of the Alliance is to provide a forum for statewide communication, advocacy, and the coordination of state tobacco control and prevention activities. The number one public policy goal of the ATCA is a major increase in tobacco tax rates.

The impressive membership of the ATCA steering committee includes representatives of: Alaskans for Drug Free Youth, Municipality of Anchorage, Bristol Bay Area Health Corporation, Tanana Chief's Conference, State of Alaska Division of Alcoholism and Drug Abuse, Yukon-Kuskokwim Health Corporation, KD Consulting, Nome Community Center, Alaska Native Health Board, Alaska Area Native Health Service, Alaska Health Fair, American Lung Association, Rural CAP Headstart, Alaska Dental Society, State of Alaska Sitka Teen Resource Center, Anchorage School District, Department of Health and Social Services, Division of Public Health, Alaska State Medical Association, Alaska Public Health Association, and the Alaska Council on the Prevention of Alcohol and Drug Abuse.

The Alaska Tobacco Control Program presently funds six local tobacco alliances (Juneau, Sitka, Ketchikan, Unalaska, Bethel, and Nome). The goal for FY97 is to establish a total of 20 tobacco alliances located in communities of 2,500 or more. These 20 alliances will reach 71% of the population in Alaska.

We Alaskans have a clearly defined problem with tobacco use and we can measure the adverse outcomes (see Schumacher in this issue). We have a federal-state-community partnership and a committment to deal with the problem. However no solution can be achieved without addressing the highly seductive and highly addictive nature of tobacco products. Simple guidance and education are not enough to influence them into making the "right" choice.

Reducing access to health risk products is one of the most effective measures available, and raising the cost of tobacco products through a higher excise tax is a proven method.

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The Prevalence of Tobacco Use Among Alaska Adults

Patricia Owen(1)
Diane E. Ingle(2)
Catherine Schumacher, MD, MSPH(2)

ABSTRACT

The Alaska Division of Public Health monitors the prevalence of smoking and other tobacco use among Alaska adults through the Alaska Behavioral Risk Factor Surveillance System, a telephone survey performed in cooperation with the U.S. Centers for Disease Control and Prevention (CDC). A total of approximately 1530 interviews are completed each year in Alaska by specially trained interviewers.

The prevalence of smoking among Alaska adults in 1994 (28.9%) was second highest in the U.S. Alaska Natives have higher smoking rates (42.9%). Overall, smoking rates have declined in the last three decades nationally. However, in recent years, little change has been found in the prevalence of smoking among adults. Alaska's rate of smokeless tobacco use has also been higher than the national rate of use. The majority of Alaska smokers (83.7%) began smoking between 10 and 20 years of age. In 1994, an estimated 121,000 Alaska adults aged 18 and older were current smokers.

INTRODUCTION

Because tobacco is one of the leading preventable causes of death and disease in the U.S. and in Alaska, the Alaska Division of Public Health monitors the prevalence of smoking and other tobacco use in Alaska through the Alaska Behavioral Risk Factor Surveillance System (BRFSS). The Alaska Division of Public Health implemented the BRFSS in 1990 with a point-in-time survey and has collected data continuously since January 1991. The survey is performed in cooperation with the U.S. Centers for Disease Control and Prevention (CDC) and gathers state based information about health related behaviors of Alaska adults through an ongoing telephone survey.

We report information from the Alaska BRFSS about smoking and other tobacco use among Alaska adults.

METHODS

Alaska is one of the 50 states or territories participating in the nationwide BRFSS. Specially trained interviewers conduct 128 interviews each month using a standard BRFSS questionnaire. Interviews are conducted over the telephone using randomly selected telephone numbers. Respondents are selected from among the adult members of the household (age 18 years and older). A total of approximately 1530 interviews are completed each year in Alaska. Data are analyzed by the CDC and the Alaska Division of Public Health, and are weighted to adjust the survey sample to represent the state adult population. Alaska uses a stratified sampling design, in which the state is divided into four regions.

The BRFSS does not include persons living in institutions, such as dormitories, barracks or nursing homes. In addition, households without telephones are excluded. Telephone coverage is about 92% in Alaska although coverage varies by region (1).

Current smokers are defined as those who have ever smoked 100 cigarettes and who smoke now. In this report, we use the 1994 BRFSS data unless otherwise stated (2).

RESULTS

1994 Results

The prevalence of smoking among Alaska adults in 1994 (28.9%) was second highest in the U.S.; Nevada had the highest prevalence (29.1%). The national range was 15.0%-29.1%, with a median of 22.6% (3). Men were more likely to be smokers than were women (31.8% of men versus 25.7% of women). The age group with the highest smoking prevalence rates (37.6%) is 18-24 years.

Married persons were less likely to be smokers than persons who were divorced, widowed or never married (Table 1). Education was strongly related to smoking status (Table 2). Only 10% of college graduates reported smoking as compared to 44.5% of those with less than a high school education. Employment is also related to smoking; individuals who reported being out of work were most likely to be smokers (Table 3).

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(1) BRFSS Coordinator, Section of Community Health and Emergency Medical Services, AK Division of Public Health, PO Box 110616, Juneau, AK 99811-0616.

(2) AK Division of Public Health, Section of Epidemology, PO Box 240249, Anchorage, AK 99524.

Table 1.
Smoking Prevalence by Marital Status

Marital Status	% Current Smokers
Married	23.4
Divorced	35.6
Widowed	34.8
Never Married	39.7
Unmarried couple	38.2

Table 2.
Smoking Prevalence by Education

Level of Education	% Current Smokers
Some high school or less	44.5
High school graduate or GED	37.3
Some college or technical school	31.1
College graduate	10.2

In Alaska, persons living in the bush area were more likely to be smokers (Figure 1).

Smoking Prevalence among Alaska Natives

Alaska Natives had higher smoking rates than the state rate (42.9% versus 26.6%)(1991-1993 BRFSS). BRFSS data showed that 46.5% of Alaska Native men smoke and 39.3% of Alaska Native women were current smokers.

Table 3.
Smoking Prevalence by Employment Status

Employment	% Current Smokers
Employed	28.1
Out of work	41.1
Homemaker	25.5
Student	28.6
Retired/unable to work	28.5

Table 4.
Alaska Adults Who Have Quit Smoking

Smoking Status	%
Ever smoked 100 cigarettes	55.9
Quit smoking	48.2*
Quit in past year	30.9**
Quit 1-15 years ago	46.6**
Quit over 15 years ago	22.5**

* Among those who ever smoked
**Among those who quit smoking

Trends in Alaska Smoking

In Alaska, smoking rates have declined. In 1982, a point-in-time survey found that 37% of adult Alaskans were current smokers, including 40% of men and 35% of women (4). The decline in smoking in Alaska corresponds to a national decline (5). In recent years, the BRFSS has shown little change in the prevalence

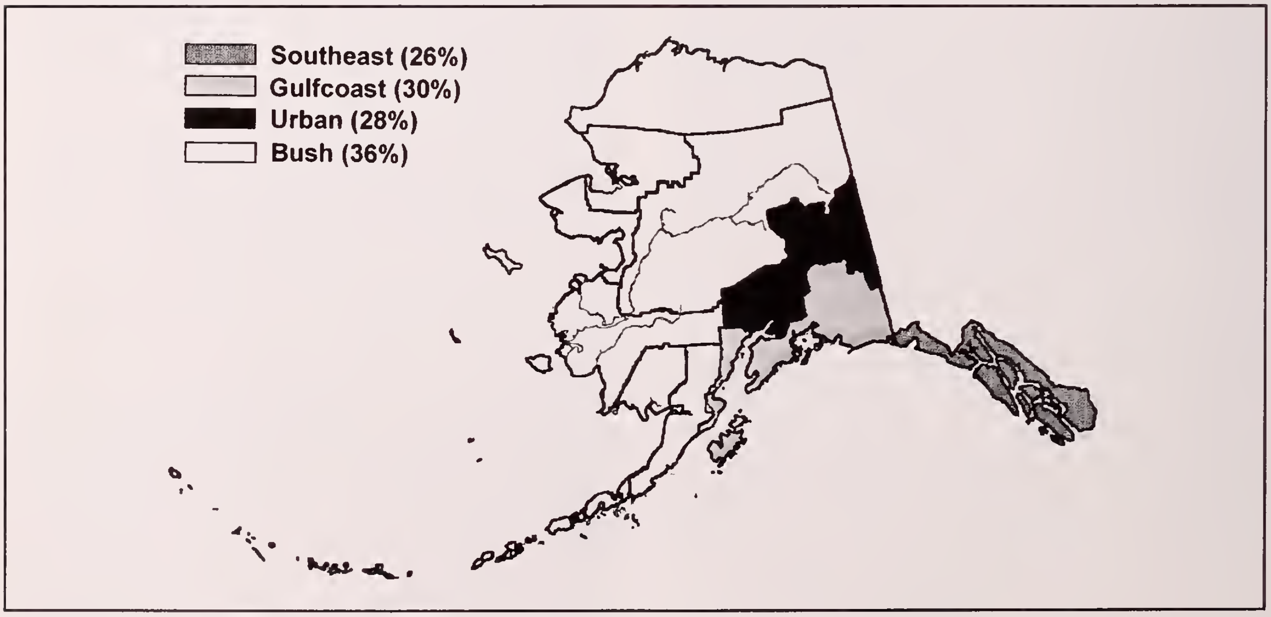

Figure 1: Smoking Prevalence in Alaska by Region: 1994 Behavioral Risk Factor Survey.

of smoking among adults. In 1991, 1992, 1993 and 1994 the prevalence of smoking was 26.0%, 28.0%, 25.8% and 28.9%,[1] respectively.

Smoking Practices

About one-half of Alaskans who have ever smoked have quit (Table 4). Among current smokers, 74.0% reported they would like to stop smoking (1993 BRFSS data). In 1994, half of all Alaska adults who smoked had tried to stop for at least 1 day in the past year.

The majority (83.7%) of Alaska smokers began smoking between 10 and 20 years of age (6). Almost 20% of those who have smoked in the past 30 days smoke more than 1 pack per day (Table 5).

Smokeless Tobacco

Current use of smokeless tobacco (chewing tobacco or snuff) was reported by 9.5% of Alaska men and 1.3% of women. Alaska's rate of smokeless tobacco use was higher than the national rate (5), based on 1991 data (Table 6). Use of smokeless tobacco products was higher among Alaska Natives (11.3%) than among the state as a whole (8.4%; 1991-93 BRFSS). The prevalence of smokeless tobacco use was highest in the Bush area (14.0% of men and 9.5% of women).

DISCUSSION

An important Year 2000 Health Objective for the Nation is to reduce cigarette smoking to a prevalence of no more than 15% of people aged 20 and older (7). In Alaska, 28.6% of adults aged 20 and older are current smokers, almost twice the Year 2000 Objective. Clearly, Alaska has a long way to go.

Alaska has one of the highest smoking prevalence rates in the United States. Alaska's smoking rates are similar to those found in Nevada and in the tobacco-growing states(5). Alaska Natives have even higher smoking rates. An estimated 121,000 Alaska adults aged 18 and older are current smokers.[2]

Smokeless tobacco use (chewing tobacco and snuff) is also higher in Alaska. The consumption of smokeless tobacco has been increasing in the United States, and most new users are adolescent boys (10,11,12). Smokeless tobacco is a major risk factor for oral cancer (13). Additionally, smokeless tobacco products contain nicotine, and their use can support nicotine addiction and may lead to cigarette use.

Almost all Alaska smokers, as well as U.S. smokers, began smoking before 20 years of age. Efforts to decrease tobacco use in the U.S. are being directed towards school-age children and adolescents , including limiting advertising and access to cigarettes (14). Additional tobacco control efforts include developing and enacting strong policies for clean indoor air, increasing excise taxes and increasing educational efforts.

Table 5.
Cigarette Consumption by Alaska Smokers

Packs per Day Smoked	Percent*
1/2 or less	29.8
More than 1/2 to 1	51.0
More than 1	18.9

*Among those who have smoked in past 30 days

Table 6.
Current Use of Smokeless Tobacco
Comparison of Alaska and U.S.,1991

	Percent Current Users	
	U.S.*	Alaska**
Men	5.6%	9.5%
Women	0.6%	0.9%

* National Health Interview Survey (5)
**Alaska Behavioral Risk Factor Surveillance System, 1991 data.

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(continued on page 51)

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[1] The data for 1994 include people who smoke irregularly (about 1%).
[2] Using 1994 population age 18 and older and smoking prevalence of 28.9%.

Tobacco Use in Rural Alaska and the Trampling Tobacco Project

Darleen N. Beltz(1)

Unlike in the "lower 48 states," where tobacco was originally grown and has been used ceremoniously by Native Americans since ancient times, tobacco has a relatively recent history among Alaska Natives (see Fortuine and Reece in this issue).

What historians find particularly striking about tobacco use in Alaska Native communities is the use of tobacco by children, some of whom are very young. Tobacco has been used on teething children to alleviate the child's discomfort. Alaska Native people have stated that they did not know that tobacco was unhealthy, and that they wouldn't have used it had they known. Within their communities, tobacco has been so accepted that its use has not been questioned.

A traditional practice of some of the Native cultures is to have a potlatch for a member of the clan who has died. At this potlatch food and other items such as tobacco are be given to the invited guests. Another tradition was the burning as an offering food and other things liked by the individual who had died. By including tobacco, it became accepted and honored.

Reincarnation is a belief of some of the Alaska Native cultures. If a child has been named after a loved one who has died, the child is considered to be the reincarnated individual. The child may be offered foods that the person liked, because they feel the child is asking for it. In the past it might have been a favorite food, such as berries or fish. Tobacco has crept into this part of the culture such that if the deceased individual had used tobacco, it, too, would be offered to the child.

Some time after its introduction, tobacco began to take on a ceremonial value in some Alaska Native groups as a gift at potlatches or other ceremonial events. Today, such ceremonial use continues in some communities.

Despite the fact that tobacco use has long been a cultural norm in most Alaska Native communities, concern about health effects has been growing in recent years. The good news is that more and more Alaska Natives are expressing a desire to quit tobacco, to avoid exposure to second-hand smoke, and to help prevent children from becoming addicted to nicotine.

The Alaska Tobacco Control Alliance (ATCA) is a group of Alaskan people concerned about the use of tobacco in their communities and their state. Members of ATCA come from the Indian Health Services, Alaska Office of Health Promotion, American Lung Association, American Cancer Society, American Heart Association, the Native regional health corporations, tribal organizations, private for-profit organizations, private non-profit organizations, and individuals. It is from this group that the Trampling Tobacco Project developed. The Project is funded by a grant from the Robert Wood Johnson Foundation specifically to reach Alaska's rural people. The Centers for Disease Control also support Northwest Portland Indian Health Board in its proposal to work with Alaska in developing clean indoor air policies in Alaska Native rural communities.

The Alaska Native people, as other indigenous people, have been negatively affected by the infiltration of the beliefs of other cultures and epidemics. This is exemplified by "The Great Death," which refers to the 1900 Alaska influenza epidemic. This epidemic killed up to 60% of Eskimo and Athabascan people. The loss of so many people disrupted Native communities, leading to a generation of people born out of great suffering, confusion, desperation, heartbreak and trauma. The world of Native people had collapsed. Their way of life was in question and their medicine men had not been able to conquer the disease.

This set the stage for modern Natives to develop an attitude which some still hold. For example, not to talk about death or to act as if it had never happened. Today this attitude is the way some deal with disease and death. This new Native questioned his own way of life and embraced the new culture, abandoning his own. They were willing to be a part of the new world and to be lead.

Today, Native people are seeing the value of their past way of life and are bringing back some of the traditions to fit into the world they now live in. They are taking back control of their lives, but some beliefs are still present that will have to be changed.

In May 1995 rural Alaska Natives from around the state met in Anchorage and organized a group called STUN (Stop Tobacco Use Now). They addressed three areas: 1) sharing concerns about tobacco use: how tobacco use is a problem in their community; 2) attitudes, values, customs, and practices that contribute to tobacco use; and 3) what can be done to reduce tobacco use. The strategic plan which was developed addresses six aspects of the tobacco issue: 1) norms and social attitudes; 2) psycho-social dynamics; 3) cultural; 4) confrontational issues; 5) tribal leadership and clergy support and 6) education.

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(1) Technical Advisor for Tobacco Control, Alaska Native Health Board, 1345 Rudakof Circle, Anchorage, Alaska.

At the meeting, STUN members discussed the difficulty of approaching elders on tobacco use. Some stated that they felt guilty in asking people to give up tobacco because there are other substances that the Native people are being asked to give up that are perceived as being even more destructive to the Native way of life than tobacco, such as drugs and alcohol.

The STUN members stated that not only did many of the Native people not realize that tobacco was a health risk, some did not know that tobacco causes cancer. People were not intentionally hurting their children by offering them tobacco. Tobacco had become a part of the community, therefore it was important that action be taken at the community level. The norm would have to be changed by informing people and encouraging all leaders to be positive role models.

Even if leaders used tobacco, they should not use tobacco in the presence of children, and should discourage youth from smoking and chewing, limit sales of tobacco to children and develop clean indoor air policies.

Many of the health providers in the community also use tobacco because it has been socially accepted. The members stated that they need to be able to develop support programs to assist the providers and other community members in quitting.

A STUN member stated that Native people learn by watching, so when the time comes for a child to act, he/she does it correctly the first time. A concern for Native people in dealing with smoking cessation classes is that they should be able to succeed the first time. If they fail, they feel discouraged and are unlikely to try again.

The members stated it was important for them to continue to combat tobacco use in their communities and agreed to continue to stay in contact with each other. The members have been holding monthly teleconferences, brainstorming ideas as to what they could do to reach their community leaders. They agreed to develop a video from each region, and to combine the segments into one and make it available to all. The video will include testimonials of persons suffering from tobacco-related diseases and of persons who have tried to quit, both successfully and unsuccessfully. A resource packet has been developed and has been sent out to 150 communities to assist these and other anti-tobacco advocates in their work against tobacco use. Forty members of STUN have been trained in the "Community Oriented Tobacco Project." This curriculum, developed by rural Alaska Native communities for rural people, involves the school, a community member, and a tobacco coordinator who work together with the community in educating and motivating people to combat tobacco use. The Trampling Tobacco Project also sponsors an Iditarod musher, supports tobacco control PSAs and media events, and provides mini-grants for community tobacco control projects.

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TRAMPLING
TOBACCO
A special project of the Alaska Native Health Board
and the Alaska Tobacco Control Alliance

Logo for Trampling Tobacco Project designed and made by Anne Marie Holen, Alaska Native Health Board.

Tobacco Use Among Alaska Youth

Michele A. Hansen, MPH, CHES(1)
Catherine Schumacher, MD, MSPH(1)
Diane E. Ingle(1)
John P. Middaugh, MD(1)
Helen L. Mehrkens, BS, CHES(2)

ABSTRACT

The Youth Risk Behavior Survey (YRBS) is a national school-based survey used to monitor health risk behaviors that contribute to the leading causes of mortality, morbidity and social problems among youth and adults in the United States. Tobacco use is one of the behaviors monitored. Both high school and middle school surveys were administered to a representative group of Alaska high school and middle school students for the first time in 1995. Surveys were administered in a confidential and anonymous manner, taking care to assure student privacy. A total of 1,634 high school students and 1,265 middle school students completed surveys.

The survey found that Alaska high school students have smoking rates higher than the national rate and that Alaska Native youth have even higher smoking rates. Furthermore, smoking is not uncommon among Alaska middle school students.

Among high school students, 36.5% were current smokers (had smoked in the past 30 days) and 21.1% had smoked on 20 or more of the previous 30 days. Boys were more likely than girls to report having used chewing tobacco or snuff in the 30 days prior to the survey (23.5% of boys and 6.7% of girls). Smokeless tobacco use increased with grade level so that 29.1% of high school senior boys had used smokeless tobacco products within the previous 30 days. Over 60% of Alaska Native students reported smoking in the previous 30 days, 43.7% reported smoking 20 or more of the previous 30 days and 22.5% reported using chewing tobacco or snuff in the previous 30 days. Over half of middle school students reported having tried smoking at least once; about one-fourth smoked at least one day in the past 30 days and 5.6% smoked on 20 or more of the past 30 days.

INTRODUCTION

The Youth Risk Behavior Survey (YRBS) was implemented by the Alaska Division of Public Health and the Alaska Department of Education in 1995 in cooperation with the National Centers for Disease Control and Prevention (CDC) (1). The YRBS is a national survey developed by the Division of Adolescent and School Health at CDC in collaboration with 71 state and local departments of education and 19 federal agencies. The survey is a component of a larger national effort to assess priority health risk behaviors that contribute to the leading causes of mortality, morbidity and social problems among youth and adults in the United States. The YRBS survey examines six categories of adolescent behavior: behaviors that result in unintentional and intentional injuries; tobacco use; alcohol and other drug use; sexual behaviors; dietary behaviors, and physical activity. We report information from the Alaska YRBS about smoking and other tobacco use among Alaska youth.

METHODS

The YRBS was administered to a sample of Alaska high school (grade 9-12) and middle school (grades 7-8) students during the spring of 1995. The high school survey consisted of 84 multiple choice questions; the middle school of 54 multiple choice questions. Students filled out the surveys during regular class time.

All public schools in Alaska with students in grades 9-12 for the high school survey and students in grades 7-8 for the middle school survey were eligible to be selected in the sample. Special education and English as a second language classes were excluded at the classroom level. Group home, correspondence and correctional schools were also excluded from the sample. A sufficient number of students were selected to give a ±5% margin of error for each question.

A two-stage sample design was used. In the first stage sampling, schools were selected from all public schools at the high school and middle school level in proportion to their enrollment size. For the second stage

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(1) Alaska Division of Public Health, Section of Epidemiology, P.O. Box 240249, Anchorage, Alaska 99524.

(2) Alaska Department of Education, Div of Teaching and Learning Support, Juneau, Alaska 99801-1894.

sampling, classes of a required subject or a required school period were randomly selected. All students in the selected classes were eligible to participate in the survey. Students, parents, schools and school districts had the opportunity to decline participation.

Teachers were given a script to read to students which established guidelines for student privacy and anonymity and the importance of the survey. Each student was given an unmarked envelope in which to seal his or her survey before turning it in. These survey envelopes remained sealed until received at a central state collection site.

The state survey was analyzed by the CDC and Westat, Inc, a CDC contractor. Analysis included the scanning of the surveys and performance of extensive edit checks to identify survey inconsistencies. The data were weighted to adjust the survey sample to represent the state youth population. The weighted data make it possible to generalize the results to all Alaska middle and high school students defined by the sample.

At the same time that Alaska implemented the YRBS, a national YRBS was conducted at the high school level. At the time of this report, the 1995 national results are still being analyzed. Therefore, the report provides the 1993 national high school results as a comparison (1). A national YRBS at the middle school level has not been done. Therefore, national comparisons are not available for the middle school results.

RESULTS

Participation

At the high school level, 38 schools were selected. Of the 38 schools, 31 chose to participate in the survey resulting in a school response rate of 82%. Within the schools that participated, the student response rate was 78%, resulting in 1,634 respondents (Table 1). At the middle school level, 40 schools were selected as part of the statewide sample. Of the 40 schools, 32 participated, resulting in a middle school response rate of 80%. At this level, 80% of the sampled students completed the surveys, resulting in 1,265 respondents (Table 2).

High School Results

Alaska high school students were more likely to be current smokers and frequent smokers than were U.S. students (Table 3). Smoking rates were similar for boys and girls. Alaska students who are older and in higher grades were more likely to be frequent smokers (smoking 20 or more days of the previous 30 days) (Table 4). However, almost 20% of ninth graders were frequent smokers.

Of Alaska high school students who had smoked in the past 30 days, the most common ways of obtaining cigarettes were: borrowed from someone else, someone

Table 1.
Demographic Characteristics of High School Respondents: 1995 Alaska Youth Risk Behavior Survey

		Unweighted Number	Weighted Percent
Age(years)*	<=15	597	36.5
	16-17	821	48.1
	>=18	215	15.3
Sex*	Male	821	47.6
	Female	807	52.4
Grade	9th	497	29.8
	10th	383	25.8
	11th	477	23.0
	12th	269	21.0
	Ungraded/other	8	0.4
Race	White-not Hispanic	1147	68.3
	Black-not Hispanic	87	5.2
	Hispanic or Latino	53	3.3
	American Indian or Alaska Native	184	15.0
	Asian or Pacific Islander	75	4.3
	Other	62	3.9

*numbers may not total 1,634 because of missing responses

Table 2.
Demographic Characteristics of Middle School Respondents: 1995 Alaska Youth Risk Behavior Survey*

		Unweighted Number	Weighted Percent
Age (years)	<=12	200	15.8
	13	591	46.8
	>=14	474	37.4
Sex**	Male	651	52.3
	Female	608	47.7
Grade**	7	636	50.6
	8	606	48.0
	Other	15	1.3

*the middle school questionnaire did not include a question on race/ethnicity.

**numbers may not total 1,265 because of missing responses

Table 3.

Cigarette Smoking Among Alaska and U.S. High School Students: 1995 Alaska Youth Risk Behavior Survey and 1993 U.S. Youth Risk Behavior Survey

	Total		Boys		Girls	
	Alaska 1995	U.S. 1993**	Alaska 1995	U.S. 1993**	Alaska 1995	U.S. 1993**
%* ever tried smoking	72.1	69.5	71.4	70.1	72.8	68.7
%* smoked on ≥ 1 of the past 30 days	36.5	30.5	36.4	29.8	36.5	31.2
%* smoked on ≥ 20 of the past 30 days	21.1	13.8	21.4	14.0	20.6	13.5

* Percent of all high school students
**Source: Reference 1

Table 4.
Percent of Alaska High School Students Who are Frequent Smokers* by Age and Grade: 1995 Alaska Youth Risk Behavior Survey

	%** Frequent Smokers
Age 15 or less	16.9
Age 16-17	22.1
Age 18+	27.5
Grade 9	19.7
Grade 10	17.8
Grade 11	23.1
Grade 12	24.7

*Frequent smokers = smoked on 20 or more of the previous 30 days
**Percent of all high school students

Table 5.
Usual Source of Cigarettes for Alaska High School Students: 1995 Alaska Youth Risk Behavior Survey

Usual Source of Cigarettes	%*
Borrowed	28.0
Bought at a store	26.3
Someone else bought	26.1
Some other way	12.6
Stole them	5.1
Vending machine	1.9

* % of students who smoked in past 30 days.

Table 6.
Use of Chewing Tobacco and Snuff among Alaska and U.S. Students: 1995 Alaska Youth Risk Behavior Survey and 1993 U.S. Youth Risk Behavior Survey

	%*
Alaska Boys	23.5
U.S. Boys**	20.4
Alaska Girls	6.7
U.S. Girls**	2.0

*% of all high school students who used chewing tobacc or snuff in the past 30 days
**Source: Reference 1

else bought the cigarettes, or the student purchased his/her own cigarettes (Table 5). Few used vending machines. Those aged 18 and older were more likely to buy their own cigarettes in stores, whereas younger students were more likely to borrow cigarettes. Even so, almost 12% of students aged 15 or less bought cigarettes in a store. Of students who bought cigarettes in a store in the 30 days prior to the survey, only 31% reported being asked to show proof of age.

Boys were more likely than girls to report having used chewing tobacco or snuff in the 30 days prior to the survey (Table 6). Alaska girls were more likely to use smokeless tobacco than were U.S. girls. Smokeless tobacco use increased with grade level, so that 29.1% of high school senior boys had used smokeless tobacco products within the previous 30 days. However, the data suggested that frequent use (use on 20 or more of the previous 30 days) was more common among boys aged 16 and 17 (Table 7).

Alaska Native students reported higher rates of smoking and of smokeless tobacco use than the state rate. Over 60% of Alaska Native students reported smoking in the previous 30 days, 43.7% reported smoking 20 or more of the previous 30 days and 22.5% reported using chewing tobacco or snuff in the previous 30 days (Table 8).

Table 7.

Use of Chewing Tobacco and Snuff among Alaska High School Boys by Age and Grade: Alaska 1995 Youth Risk Behavior Survey

	%* who are current users**	%* who are frequent users***
Age 15 or less	16.5	5.5
Age 16-17	26.1	8.2
Age 18+	29.8	6.6
Grade 9	19.6	7.0
Grade 10	22.0	6.5
Grade 11	25.3	8.3
Grade 12	29.1	6.2

*Percent of all high school boys

**Current use = used at least once in past 30 days

***Frequent use = used on 20 or more of the previous 30 days

Table 8.

Tobacco Use by Alaska Native High School Students: 1995 Alaska Youth Risk Behavior Survey

	%
%* who ever tried cigarette smoking	90.7
%* who smoked in the past 30 days	61.9
%* who smoked on 20 or more of the past 30 days	43.7
%* who have used chewing tobacco or snuff in the previous 30 days	22.5

*Percent of all Alaska Native high school students

Table 9.

Tobacco Use by Alaska Middle School Students: 1995 Alaska Youth Risk Behavior Survey

	%
%* who ever tried cigarette smoking	58.3
%* who smoked in the past 30 days	24.8
%* who smoked on 20 or more of the past 30 days	5.6
%* who have ever used chewing tobacco or snuff	30.3

*Percent of all Alaska middle school students (Grades 7 and 8)

Middle School Results

Over half of middle school students have tried smoking at least once; about one-fourth smoked at least one day in the past 30 days and 5.6% smoked on 20 or more of the past 30 days (Table 9). The results are similar for boys and girls, with the exception of smokeless tobacco use. Boys are more likely to report ever having used chewing tobacco or snuff than are girls (37.3% versus 22.1%).

The most common way middle school students obtained cigarettes is by borrowing them from someone else (37.2% of smokers); very few middle school smokers reported purchasing cigarettes at a store or vending machine.

DISCUSSION

Alaska adults have one of the highest smoking prevalence rates in the United States, similar to those found in Nevada and in the tobacco-growing states (2). The YRBS results indicate that Alaska high school students also have smoking rates higher than the national rate. Alaska Natives have even higher smoking rates. Furthermore, smoking is not uncommon among Alaska middle school students.

Smokeless tobacco use (chewing tobacco and snuff) is also used commonly by Alaska high school boys. The consumption of smokeless tobacco has been increasing in the United States, and most new users are adolescent boys (3,4,5). Smokeless tobacco is a major risk factor for oral cancer (6). Additionally, smokeless tobacco products contain nicotine, and their use can support nicotine addiction and may lead to cigarette use.

The majority of Alaska smokers (83.7%), began smoking between the ages of 10 and 20 years (7). Tobacco prevention education within schools is one of many interventions that can be effective at preventing tobacco use among youth. A 1994 Alaska School Health Education Profile survey conducted by the Alaska Department of Education found that 83% of secondary school principals reported that health education was a graduation requirement in their school (8). However 61% also reported that students were only required to take one health class from grade 6 through graduation.

Health education theories often are based on the premise that behavior change involves a certain level of knowledge about a behavior, attitudes that are supportive of the desirable behaviors and having the skills necessary to use the desirable behaviors. The School Health Education Profile found that 90% of health teachers teaching tobacco use prevention reported teaching to increase students' knowledge, 87% taught to improve students' attitudes and 76% taught skills to increase healthy behaviors (8).

An important Healthy People 2000 objective is for no more than 4% of boys age 12-24 years to be current users of smokeless tobacco (9). Although the YRBS only measures youth who are in school, Alaska's estimate of 23% among high school boys indicates that we have a long way to go.

The statewide YRBS provides descriptive data on the *what, who, where* and *when* of tobacco use among Alaska students. The questions of *why* and *how* cannot be answered by this survey. However, the YRBS for the first time provides our state with baseline data that is comparable to the nation.

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Tobacco Erases 30 Years of Progress:

Preliminary analysis of the effect of tobacco smoking on Alaska Native birth weight

Neil J. Murphy, MD(1)
Steve W. Butler, MS(2)
Kenneth M. Petersen, MD(3)
Victorie Heart, RNC, MS(4)
Colleen M. Murphy, MD(1)

ABSTRACT

Objective: Investigate the relationship between tobacco and/or alcohol use and Alaska Native birth weight.

Methods: Data on weight, tobacco smoking and alcohol use among Alaska Natives were abstracted from 1989-91 Indian Health Service natality records based on birth certificates.

Results: Birth certificate data were available for 9,175 live births. Single live births were analyzed for 8,994 Alaska Natives. In women with no tobacco smoking the mean birth weight of their infants was 3,571 g; 1-5 cigarettes/day 3,429 g; 6-10 cigarettes/day 3,332 g ($p < .05$); and > 10 cigarettes/day 3,260 g ($p < .05$). Infants of Alaska Natives who reported no alcohol and no tobacco use had a mean birth weight of 3,579 g; alcohol use but no tobacco use 3,452 g; no alcohol but tobacco use 3,388 g; and both alcohol and tobacco use 3,281 g. ($p < .001$)

Conclusions: The mean birth weight of infants born to Alaska Native women with the highest use of tobacco were reduced by over 300 g compared to non-smoking Alaska Native women. Mean infant birth weight of tobacco smoking Yup'ik women in 1989-91 were reduced by over 400 g, comparable to weights reported in the 1960s.

INTRODUCTION

In 1960-62 a cohort study in Southwest Alaska revealed a profile of birth weight and infant mortality similar to a third world country with low birth weights, and infant mortality four times greater than that of U.S. whites (1). In 1980, 20 years after the original study, mean birth weight and infant mortality were similar to that of U.S. whites, due in part to improved health care and immunizations (2).

Maternal tobacco smoking during pregnancy has been shown to decrease infant birth weight, both with maternal smoking (3-5) and exposure to environmental tobacco smoke (ETS) (6,7). Birth weight is further decreased with the combined use of tobacco and alcohol (8). As reported in this journal and elsewhere in the literature, tobacco smoking and ETS have been associated with increased pediatric respiratory disease (7, 9) and sudden infant death syndrome (10).

This study reports the relationship of tobacco smoking and alcohol use and Alaska Native birth weight.

METHODS

This review of single Alaska Native live births is based on Indian Health Service natality information. The data set is derived from State of Alaska Bureau of Vital Statistics birth certificate data for 1989, 1990, and 1991.

The Yup'ik Inuit predominantly comprise the population reported for the Yukon Kuskokwim Delta Service Unit; Inupiaq Inuit in Kotzebue, Barrow, and Norton Sound Service Units; Athabascan Indian in Interior Service Unit; Tlingit, Haida, and Tsimshian are included as Southeast Coastal Indians from Mt. Edgecumbe and Annette Island Service Units; and Aleuts are included in the mixture of Alaska Natives at the Alaska Native Medical Center (ANMC). Tobacco smoking and alcohol intake were recorded by the birth attendant from

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(1) Alaska Area Native Health Service, Alaska Native Medical Center, Department of Obstetrics and Gynecology, Anchorage, Alaska 99501
(2) Alaska Area Native Health Service, Division of Planning, Evaluation and Health Statistics
(3) Alaska Area Native Health Service, Alaska Native Medical Center, Department of Pediatrics
(4) Alaska Area Native Health Service, Maternal Child Health Coordinator

prenatal records without regard to trimester of use. Smokeless tobacco use was not recorded during the years reported.

Mean weights for each category were compared using a two sided t-test with $p < .05$ for statistical significance.

RESULTS

The total number of Alaska Native births reviewed was 9,175 (3021 for 1989, 3099 for 1990, and 3055 for 1991). Of the 8,994 Alaska Native single live births, there were 4,093 female and 4,195 male births of 20 or more weeks gestation. The overall mean birth weight was 3,501 g. The mean birth weights by race were: Southeast Coastal Indian 3,581 g (n = 1,171, SE 16.7)($p < .05$); Athabascan Indian 3,489 g (n = 902, SE 18.0); Yup'ik Inuit 3,471 g (n = 1,879, SE 13.2); Inupiaq Inuit 3,468 g (n = 1,763, SE 13.5); and ANMC 3,475 g (n = 3,262, SE 9.9).

Tobacco smoking status was reported in 8,435 women. For all Alaska Native births of women who reported no tobacco smoking the mean birth weight was 3,571 g (n = 5,477, SE 7.38); 1-5 cigarettes/day 3,429 g (n = 1,025, SE 16.4); 6-10 cigarettes/day 3,332 g (n = 1,324, SE 15.2, $p < .05$); and > 10 cigarettes/day 3,260 g (n = 609, SE 24.5, $p < .05$) (Figure 1.)

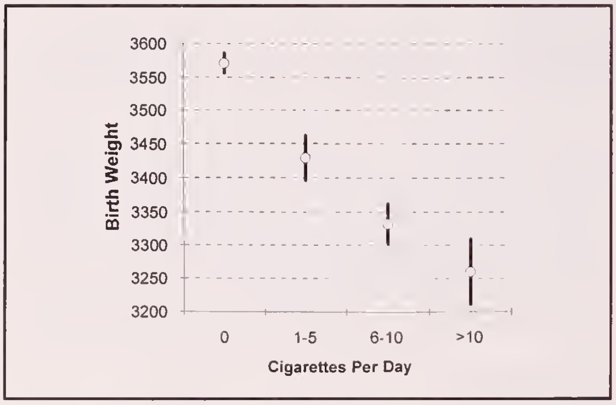

Figure 1. 1989-1991 Alaska Native mean birth weight in grams by number of cigarettes per day (bar indicates 95% confidence interval)

For Yup'ik Inuit who reported no tobacco smoking the mean birth weight was 3,512 g (n = 1,360, SE 14.6); 1-5 cigarettes/day 3,464 g (n = 116, SE 48.5); 6-10 cigarettes/day 3,248 g (n = 86, SE 74.4, $p < .05$); and > 10 cigarettes/day 3,110 (n = 25, SE 132.4, $p < .05$) (Figure 2).

All Alaska Native births were categorized by use of alcohol as well as tobacco. Among Alaska Native women who reported no alcohol and no tobacco use the mean birth weight was 3,579 g (n = 5,632, SE 7.1); alcohol use but no tobacco 3,452 g (n = 4,864, SE 8.9, $p < .001$); noalcohol but tobacco use 3,388 g (n = 5,120, SE 7.5, $p < .001$); and both alcohol and tobacco use 3,281 g (n = 5,376, SE = 7.7, $p < .001$) (Figure 3).

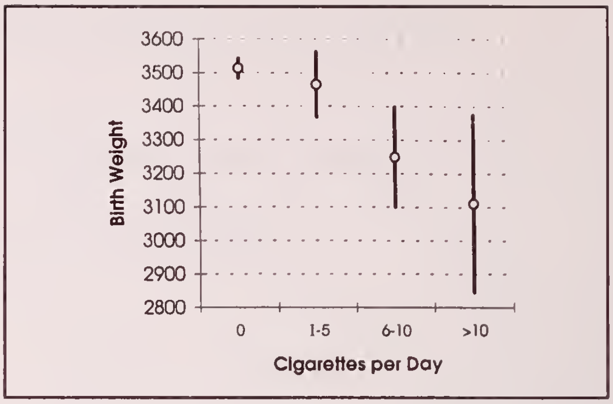

Figure 2. 1989-1991 Yup'ik Inuit mean birth weight in grams by number of cigarettes per day (bar indicates 95% confidence interval)

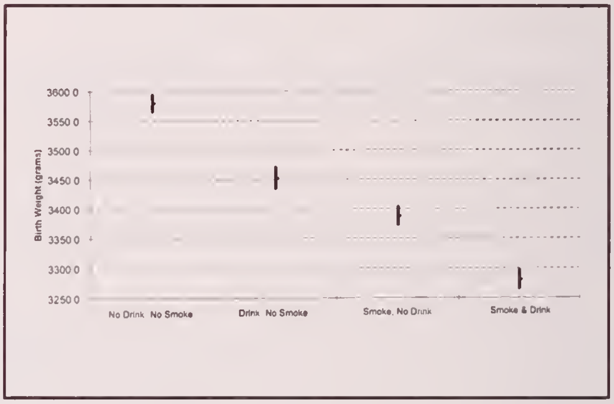

Figure 3. 1989-1991 Alaska Native mean birth weight in grams by tobacco smoking and use of alcohol (bar indicates 95% confidence interval)

DISCUSSION

The mean birth weight of infants born to tobacco smoking Alaska Native women was lower than the mean birth weight of infants born to non-smoking Alaska Native women. Women who reported the highest use of tobacco had infants whose weights were 300 or more grams lower. The mean 1989-91 birth weights of tobacco smoking Yup'ik women were lower than the mean birth weight for non-smoking Yup'ik women by over 400 g. The mean 1989-91 birth weights of tobacco smoking Yup'ik women was similar to the birth weights reported in 1960-62, when the infant mortality in the Yup'ik was 4 times that of U.S whites (1,2).

The observed lower birth weights with maternal tobacco smoking among Alaska Native mirrors that reported in North American Natives (3), as well as other populations (4,5). Decreased birth weight has also been reported with ETS. (6,7) The mean Alaska Native birth weights were also observed to be significantly lower with the combined use of alcohol and tobacco, as previously reported in Denmark (8). As shown in this journal and elsewhere in the literature, maternal smoking and

ETS have been associated with increased pediatric hospitalization for respiratory disease (7, 9) and sudden infant death syndrome (10). These data are more chilling for the future when one considers that Alaska Natives currently have one of the highest rates of smoking in North America (11).

The mechanism of decreased birth weight in maternal tobacco use appears in part to be associated with vascular constriction and decreased uteroplacental blood flow (12-15). Cotinine serum levels have been quantitatively associated with decreased mean fetal birth weight (16). Ultrastructual changes in placental villi and fetal capillaries have been documented in smoking mothers and hypoxia, nicotine and carbon monoxide have been shown to pass the placental barrier (17).

Though both alcohol and tobacco use are preventable causes of decreased birth weight, these data confirm that smoking makes a quantitatively larger contribution (3). Public health measures in Alaska should concentrate on elimination of maternal tobacco and alcohol intake, as well as exposure to passive smoking, during pregnancy.

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Association between Maternal Smoking and Severe Respiratory Syncytial Virus Infections and Sudden Infant Death Syndrome

Rosalyn Singleton, MD(1)

RESPIRATORY SYNCYTIAL VIRUS

Studies have clearly demonstrated that maternal smoking is associated with increased risk of lower respiratory illness (LRI) in the first few years of life. Respiratory syncytial virus (RSV) is the most common cause of lower respiratory illness, usually bronchiolitis or pneumonia, in infants and young children. Respiratory syncytial virus-related LRI is responsible for 100,000 hospitalizations and 5,000 deaths in the U.S. annually (1). In Alaska Natives, rates of RSV-associated hospitalization for children less than one year of age are 2 to 50 times the rates reported in other U.S. populations (2).

Passive smoke exposure has been shown to increase airway hyperresponsiveness in young infants and is related to persistent wheezing in grade school asthmatic children. In a case control study by McConnochie and Roghmann, any passive smoking and maternal smoking were statistically significant predictors of bronchiolitis (3). In a case control study by Duff et al, comparing actively wheezing children under two years of age to children without respiratory symptoms, a larger proportion of wheezing patients than controls were exposed to tobacco smoke, and most of the smoke-exposed children had cotinine levels (> 10 ng/ml) suggestive of heavy smoke exposure (4). A threshold level at which passive smoke exposure becomes clinically significant in causing or aggravating airway hyperresponsiveness has not been defined. Higher rates of asthma and increased usage of asthma medications have been documented in children exposed to mothers who smoke at least one-half pack of cigarettes per day. McConnochie and Roghmann found that maternal smoking was a powerful predictor of wheezing even in older children up to age 13 (5).

SUDDEN INFANT DEATH SYNDROME

A population-based study conducted in Sweden assessed risk factors for sudden infant death syndrome (SIDS). All infants surviving the first week of life were included (n = 279,938). The overall rate of SIDS was 0.7 per 1000 first-week survivors; maternal smoking doubled the risk. There was a clear dose-response relationship. It is estimated that in some developed countries smoking may be the single most important preventable risk factor for sudden infant death syndrome (6).

Maternal smoking was established as a risk factor in a U.S. study published in 1993 that compared 485 SIDS cases with 1,800 control infants. Results showed that the risk of succumbing to SIDS was four times higher for babies whose mothers smoked during pregnancy than for those whose mothers did not (7). Infants of mothers who smoked after the baby's birth also had a greater risk of SIDS, as did infants in households where the father or another family member smoked, and the increased risk was dose related (8).

In the 1988 National Maternal and Infant Health Survey, review of a number of demographic, prenatal, and environmental factors showed that, among characteristics generally thought to be risk factors, only maternal smoking during pregnancy was independently associated with SIDS. Data from this nationally representative sample indicate that if women refrained from smoking while pregnant, up to 30% of SIDS might be prevented (9).

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(continued on page 51)

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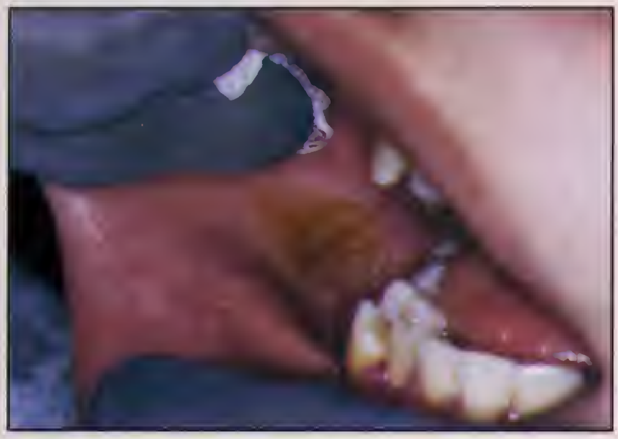
(1) Alaska Area Native Health Service, 250 Gambell Street, Anchorage, Alaska 99501.

Smokeless Tobacco and Oral Disease

James C. Singleton, DDS, PC[1]

The oral effects of smokeless tobacco are numerous. They include: oral and esophageal cancer; gum disease with early tooth loss; tooth decay; and halitosis (1). Women who do not smoke but use snuff chronically, are 50 times more likely to develop oral cancer than non-users (2). Young people are not immune from developing cancer and pre-cancerous lesions at the site of contact with the tobacco. The figure below shows marked leukoplakia in a 13-year-old Alaskan smokeless tobacco user.



Nationally there has been a dramatic increase in the use of moist snuff, especially in young people (3). Among Alaska Natives, there is widespread use of cigarettes and smokeless tobacco by both children and adults which exceeds the national average. Smokeless tobacco use among Alaska Native children has been reported in 13% of kindergarten children, increasing to 30% in high school (4). Results of a more recent study indicate that as many as 68% of children 7-11 years old reported they had used snuff in the past and as high as 52% had used it recently (5). There was no statistically significant difference in use between boys and girls. Use increased with age and positive correlations were found with peer and family use. When these same children were questioned, 96% said they understood that smokeless tobacco products could cause oral cancer!

The Oral Health Survey conducted by the Indian Health Service in 1991 showed that in the Alaska Area the number of observable oral soft tissue changes found in school children which could be attributed to the use of smokeless tobacco was 30% when all forms of oral manifestations were included (6). Three-fourths of these lesions are reversible within a year if the habit is discontinued.

Other studies substantiate the fact that smokeless tobacco use in American Indians and Alaska Native children is considerably higher (30-40%) than the same age group in the general population (5). Rates of occasional use of smokeless tobacco by American Indians and Alaska Natives are high even among the very young—74% of the girls and 90% of the boys reported weekly use of smokeless tobacco began using it before the age of 10.

It is obvious that children are not capable of comprehending the lifelong consequences of using snuff , nor have they developed the refusal skills necessary to avoid this dangerous habit. Use by family members, peers, and community leaders encourages children to model those they look up to. Experimentation generally leads to chemical addiction. Abstinence from smokeless tobacco results in signs and symptoms of nicotine deprivation that are similar to those seen in smokers after they stop smoking. Anecdotal reports of kindergartners placing a pinch of snuff in carbonated beverages and downing it to get a caffeine enhanced nicotine buzz are perfect examples of the type of experimentation and progression to more powerful medications and combinations. This progression has lead investigators to label snuff as a "gateway " substance since it frequently leads to the use of cigarettes, alcohol, and illicit drugs.

Even more alarming is the fact that in the communities where these surveys were conducted, family and community leaders did not consider smokeless tobacco use by children to be a major health concern. Unless the concern of these individuals can be raised; the future of American Indian and Alaska Native will include poor oral health.

Recognizing the above, community oriented prevention strategies are needed. Successful programs include the development of coping and refusal skills, emphasis on recognition of short term health consequences, and the inclusion of influential community role models. As health care providers we cannot allow children to be passive participants in their own distruction. Until they reach the age where they can make informed independent decisions about their own health they must be protected and encouraged by those responsible for their care.

(continued on page 51)

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(1) Area Pediatric Dental Consultant, Alaska Native Medical Center, 250 Gambell St., Anchorage, Alaska 99501.

Nutrition and Smoking Cessation

Charlotte Stefanich, MS, RD, CHES[1]

The fear of weight gain serves as a barrier for many clients who have a desire to stop smoking. This fear is founded on common knowledge and documented changes. The average weight gain following smoking cessation is about ten pounds and 15 % of women may gain thirty pounds or more (1). The physician's challenge is to help clients manage their expectations and behaviors to keep the weight gain to a minimum.

PHYSIOLOGICAL MECHANISMS

The smoker has a higher total energy expenditure than the non-smoker. The metabolic rate increases under the influence of nicotine. Caffeine also increases this rate, so frequently smokers have two agents acting to increase their caloric expenditure. Nicotine does not seem to have an appetite suppressant effect, nor does transdermal nicotine prevent weight gain. Smokers also have increased levels of lipoprotein lipase, the enzyme which facilitates removal of lipid from the blood and entry through the capillary wall into the adipose cell. This level does not decrease immediately at cessation of smoking and may be a factor in the weight gain that is common after quitting (2).

The use of dexfenfluoramine or fluoxetine did not prevent the weight gain associated with smoking cessation. The serotonergic drugs reduced initial weight gain following smoking cessation, but when they were discontinued, weight increased. Clients had a total weight gain similar to those who had not had drug therapy (3). Helping clients reframe the weight gain as the weight they would be if they had not been a smoker has been suggested (4). It is important to emphasize that the health risks from smoking remain greater than those from additional weight .

Components in cigarette smoke are implicated in free radical mechanisms of protein damage. To compensate for this, the Recommended Dietary Allowance for ascorbic acid (Vitamin C) is forty milligrams more per day for the smoker (5). Functions of other anti-oxidants such as Vitamin E and beta carotene in damage prevention are being studied.

Smoking also influences the olfactory and taste sensations associated with food. Self-reported feelings of hunger increase during cessation even by clients who were treated with nicotine replacement. As most smokers consume more calories, alcohol and sugar but less vitamins than non-smokers, these feelings are often dealt with by eating high sugar, high fat foods.

Nicotine also decreases the lower esophageal sphincter pressure resulting in frequent bouts of heartburn or esophagitis. Tobacco cessation reduces or eliminates these symptoms although they may continue during the withdrawal period. Cutting down on acid foods, fatty foods, and caffeine may help alleviate these problems.

BEHAVIORAL CHANGES

Part of the challenge in stopping smoking is the replacement of the hand to mouth activity. It is important to alert the client about avoiding the substitution of high calorie foods for cigarettes. Clients also need to recognize that often food serves as the trigger for a cigarette. Caffeine consumption parallels cigarettes smoked. Caffeine must also be factored into the total smoking cessation experience, as caffeine withdrawal also produces symptoms.

MANAGING WITHDRAWAL

Food choices during withdrawal can alleviate symptoms or promote greater discomfort. Heartburn may be decreased with a low fat diet and increased fluid intake. Smokers should be encouraged to monitor their food choices using the USDA Food Guide Pyramid just as they monitor their smoking behavior (Figure 1). The first cup of coffee should be accompanied by a nutrient containing food rather than a cigarette. Help the client review activities and times which elicit smoking. Have them list options which can be pursued to change either place, environment, or mental attitude in those situations.

Certainly exercise should be pursued as an alternative for sedentary smokers. Adding movement will keep the energy expenditure at a higher level and promote pulmonary and cardiovascular fitness. It needs to be a component of any healthy lifestyle. Help clients identify those activities they are willing to pursue so that they will be an enjoyable part of their day. Ask questions about exercise patterns on subsequent visits to emphasize their importance. In Alaska, the smoker who plans to quit needs to identify activity options for all seasons.

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(1) Registered Dietician, Dietary Dept., Alaska Native Medical Center, 250 Gambell Street, Anchorage, Alaska 99501.

Walking is the easiest and least stressful exercise and requires little preparation. Traditional activities from berry picking to skiing should be encouraged.

POST-WITHDRAWAL NUTRITION

Relapse is a reality. Most clients attempt to quit smoking many times before they are successful. Each one of these attempts should be framed as a learning experience, rather than a failure. Certainly this is true of the client who has gained weight in initial attempts. Clients can develop better dietary and exercise patterns. Those changes may play a role in harm reduction from continued smoking.

There are many educational materials available which can be used to help guide a person's food and exercise choices. The American Cancer, Heart, and Lung Associations, the National Cancer Institute, the Cooperative Extension Agency and hospital patient education departments are some sources. Such materials increase clients' awareness of appropriate steps to take in stopping smoking. See the sample patient hand-out on this page.

For clients needing or requesting personalized dietary information connect them with a dietitian in your community. More intensive programs of intervention with group support or one-on-one counseling are also available in many places. Most of these feature healthy lifestyle changes which contain a nutrition component.

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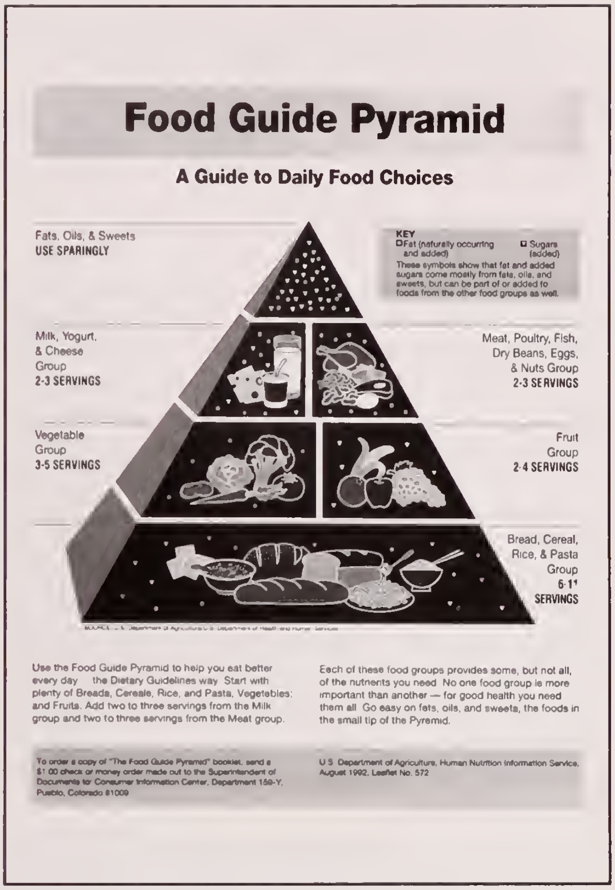
Food Guide Pyramid
A Guide to Daily Food Choices
Fats, Oils, & Sweets
USE SPARINGLY
KEY
□ Fat (naturally occurring and added)
▣ Sugars (added)
Milk, Yogurt, & Cheese Group
2-3 SERVINGS
Meat, Poultry, Fish, Dry Beans, Eggs, & Nuts Group
2-3 SERVINGS
Vegetable Group
3-5 SERVINGS
Fruit Group
2-4 SERVINGS
Bread, Cereal, Rice, & Pasta Group
6-11 SERVINGS

These symbols show that fat and added sugars come mostly from fats, oils, and sweets, but can be part of or added to foods from the other food groups as well.

Use the Food Guide Pyramid to help you eat better every day the Dietary Guidelines way. Start with plenty of Breads, Cereals, Rice, and Pasta, Vegetables; and Fruits. Add two to three servings from the Milk group and two to three servings from the Meat group.

Each of these food groups provides some, but not all, of the nutrients you need. No one food group is more important than another — for good health you need them all. Go easy on fats, oils, and sweets, the foods in the small tip of the Pyramid.

To order a copy of "The Food Guide Pyramid" booklet, send a $1.00 check or money order made out to the Superintendent of Documents to: Consumer Information Center, Department 159-Y, Pueblo, Colorado 81009

U.S. Department of Agriculture, Human Nutrition Information Service, August 1992, Leaflet No. 572

Figure 1. USDA Food Pyramid.

WHY WEIGHT ?

Be aware of when you eat and when you smoke. Does one act trigger the other?

Choose other things to do as you quit-

- Walk
- Hike
- Swim
- Join an athletic club and attend classes
- Learn a new skill

Follow the Food Guide Pyramid

Choose fruits and vegetables as between meal foods

Chew sugarless gum

Drink water daily

Cut down on coffee, tea, soda, and chocolate

Learn to relax

Increase in Lung Cancer in Alaska Natives:

How high will the rates go?

Anne P. Lanier, MD, MPH(1)

Cancer of the lung has risen dramatically among Alaska Natives in recent years. It is now THE leading cause of cancer death in Alaska Native men and women. This is a remarkable fact considering that cancer was reported to occur infrequently in this population as recently as the middle of the century. Lung cancer is now the most frequently diagnosed cancer in Alaska Native men, and ranks second in women. It is also the cancer for which the rates are increasing most rapidly. In1950, *one* cancer death in an Alaska Native was recorded to the Territory's office of vital statistics (1). Lung cancer deaths in Alaska Natives now approach 30 per year (2).

In 1969 (the first complete year of data in the Alaska Native Tumor Registry), six patients were diagnosed with lung cancer. In 1995 there were 45, yet the population increase from 1970 to 1990 was less than double (51,000 to 86,000).

Several early physicians were impressed by the low occurrence of lung cancer in the Alaska Native population and reported their findings. In the late 1950s and early 1960s, E. Fuller Torrey found only four cases of lung cancer among 170 cancer patients seen at the Indian Health Hospitals in Anchorage and Mt. Edgecumbe from 1956-1961 (3). Regarding the findings for lung cancer, he stated that "the low incidence is striking".

M. Walter Johnson reviewed cases at the Alaska Native Medical Center and found only nine patients with lung cancer diagnosed during the seven year period, 1969-73. There were five men (all heavy smokers), four women (one of whom was a smoker)(4). All four cancers in women were adenocarcinoma. Robert Fortuine reviewed cancer data for the Bethel region for the years 1957-67. Among 85 patients diagnosed with cancer at the Bethel hospital during those years, five had lung cancer, four of the five patients were men (5).

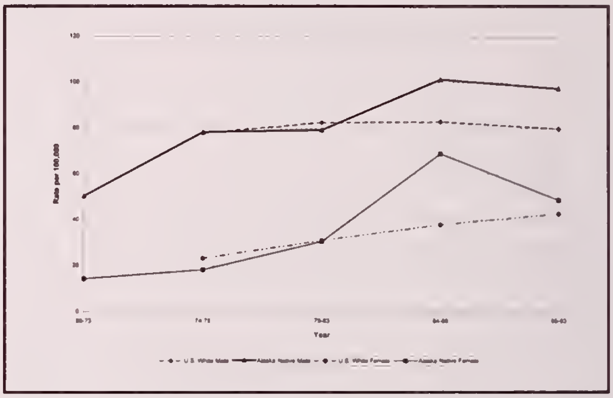

Figure 1. Incidence rates of lung cancer for men and women, Alaska Native vs. US Whites (rates age-adjusted to the US 1970 population.

Figure 1 compares incidence rates for lung cancer in Alaska Natives for the last 25 years with those of the US. Rates for AN are from the Alaska Native Tumor Registry. US data are for the White population and are taken from the National Cancer Institute's SEER (Statistics, Epidemiology and End Results) program; a nationwide system of population-based tumor registries in the US (6). The SEER program has been in existence since 1973. Cancer incidence data are not available for the non-Native population of Alaska. The figure indicates that Alaska Native rates for lung cancer were below the US in the sixties, caught up in the seventies, and now *exceed* US rates for both men and women.

Rates of deaths from lung cancer are available for a longer period of time. Figure 2 compares death rates from lung cancer for Alaska Natives and US whites for most of the 20th century. The curves for lung cancer in Alaska Natives parallel those of US whites. However, the curves are shifted to the right indicating that, compared to the US in general, the increase in lung cancer death rate began about 20 years later in Alaska Native men, and about 10 years later in women. Although there is little information about past tobacco use patterns in Alaska, reported delay of frequent regular use of to-

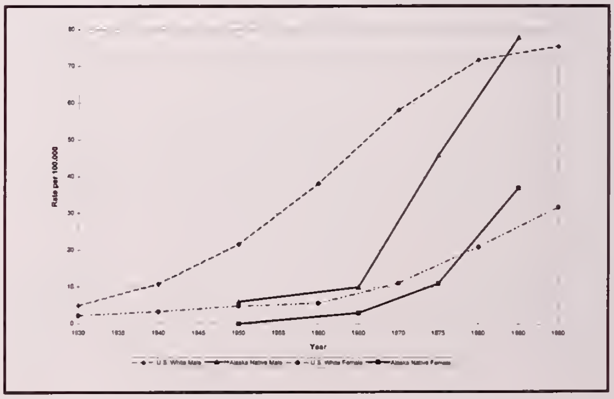

Figure 2. Death rates for lung cancer for men and women, Alaska Native vs. US (rates age-adjusted to the US 1970 population)

bacco in rural Alaska until after World War II (in contrast to after World War I in the "lower 48") would explain the delayed rise in lung cancer death. As other articles in this issue document, the prevalence of current smokers in Alaska Natives is nearly double that of the national average. In addition a large number of adults (nearly 30%) are ex-smokers. It is impossible to predict exactly when the lung cancer epidemic will peak or how high it will go, but tobacco related deaths now exceed the national average. This is particularly regretable since lung cancer and other tobacco-related deaths are PREVENTABLE.

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(1) Alaska Area Native Health Service, Indian Health Service, 250 Gambell Street, Anchorage, Alaska 99501.

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Community Service Announcement

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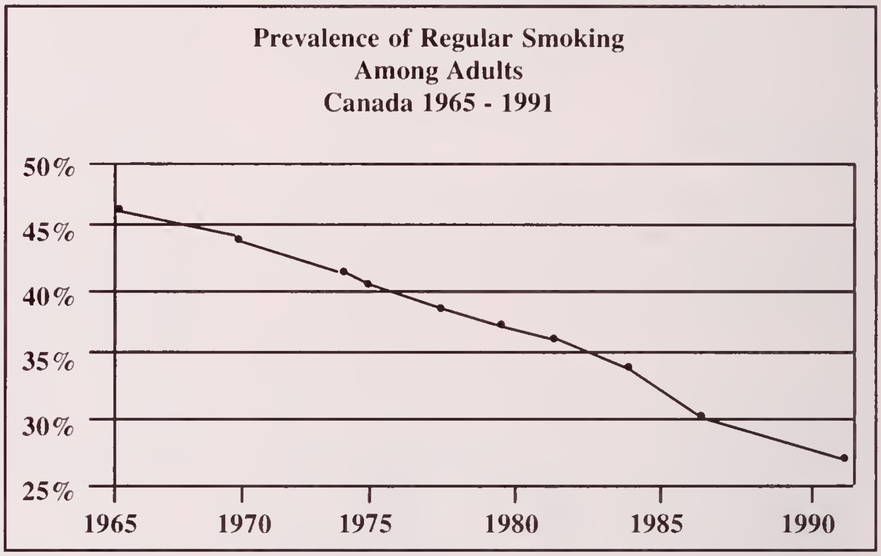
Tobacco Taxes:
The Canadian Experience

David T. Sweanor[(1)]



Tobacco control efforts in Canada are now generally considered to be among the most effective in the world. During the past decade, not only were taxes increased to among the highest in the world, but tobacco advertising was banned, prominent warnings were placed on packages, and legislated protection from environmental tobacco smoke has been implemented for a great many areas. As a result of all these measures, Canada has seen a reduction in tobacco consumption that is greater than any other major country has ever accomplished in any ten year period. There has also been an accelerating pace to this decline (see graph).

This report concentrates on taxation policy. That is not to say that other factors have not been an important part of Canada's reduction in tobacco use over the past 10 years. But the evidence suggests that, of all elements, the single most effective policy has been increasing taxes.

It is generally recognized in economic theory, as well as in every day life, that purchasing decisions are influenced by changes in the price of goods. Economists talk about the degree of responsiveness of demand to changes in price as a product's "price elasticity of demand." American research has suggested that tobacco products show a price elasticity of about -0.4 among adults. The research also indicated that young people were particularly price-sensitive. In fact, the most comprehensive look at teenagers in the United States indicated a price elasticity of -1.4. What these figures mean is that every 10% increase in the "real" (i.e., inflation adjusted) price of tobacco would reduce adult consumption by about 4% and youth consumption by about 14%. Canadian experience with raising taxes during the past decade has lead to estimates of price elasticity that are very much in line with the earlier American research.

Prevalence declines in Canada have been very significant over time, and particularly steep during the time of rising prices. An analysis of major prevalence surveys from the Canadian government shows a clear trend. Prevalence of regular smoking has fallen much more quickly as prices rose in the past 10 years. Prevalence of regular smoking among adults in Canada decreased from 46% in 1965 to 26% in 1991. The average annual decline in regular smoking up until 1981 was approximately 1.5%. Between 1981 and 1991, when taxes increased significantly, the average annual decline increased to approximately 3.2%.

The research on the effects of tobacco price increases indicated that young people were more price-sensitive than adults. The Canadian experience confirms this view. The most telling indication of price-sensitivity is shown when we juxtapose teenage smoking trends and the real price of cigarettes (see Nakamura this issue).

The relationship between price and demand is such that the tobacco tax increases, while reducing consumption, led to massive increases in tobacco tax revenue. From the standpoint of any government, the revenue gains are enhanced by the fact that taxes make up only a portion of the final selling price. Therefore an increase in the tax represents a proportionately smaller increase in overall price. This means that a government with a tobacco tax that currently represents 20% of the retail price of cigarettes could double its tax per pack while total sales would be expected to decline by only about 8%. The net revenue gain would be in the range of 84%.

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(1) Senior Legal Councel, Non-Smoker's Rights Association, 130 Albert Street, Suite 1903, Ottawa, Ontario, Canada, K1P 5G4.

The Canadian experience has been replicated elsewhere. This report is intended as a case study in using tax policy as a key element in a comprehensive tobacco prevention and control strategy. The industry knows every bit as well as the health lobby that increases in tobacco tax are the single most potent of all currently used measures to reduce tobacco consumption.

Due to cigarette smuggling caused by low U.S. tobacco taxes, the Canadian government announced a rollback in its federal tax on February 8, 1994. The rollback reduced the federal excise tax by between 50 cents and $1.00 (CD) per pack. Quebec, Ontario, New Brunswick, Nova Scotia and Prince Edward Island also reduced provincial taxes in this range.

The tax rollback was a response to growth in cigarette smuggling resulting from the tax discrepancy between Canada and the U.S. Canadian manufacturers shipped large quantities of tobacco products free of Canadian taxes to the northern United States, from where smugglers would bring them back into Canada. The fact that Canadian cigarettes are a different blend from American, and that there is very little demand in Canada for the American style means that virtually all cigarettes smuggled into Canada originated from Canada. Smuggled cigarettes represented between 20 and 30 percent of the total Canadian market, and a higher percentage in Quebec, Ontario and New Brunswick.

The tobacco industry warns that the U.S. will face a smuggling problem similar to Canada's if the U.S. raises tobacco taxes. In fact, a higher U.S. federal tax would eliminate smuggling along the Canadian border. By balancing out price discrepancies, such a tax increase would substantially reduce the small amount of bootlegging that occurs between states, between Native American reservations and states, and between military bases and states .

Alaska should not encounter a smuggling problem like Canada's if a tax increase is enacted. Alaska's population is not close to a low tax jurisdiction since the big tax reductions happened in Eastern Canada. The Western provinces refused to lower taxes and have avoided any significant smuggling problems.

This article is a synopsis of the following report:

Sweanor DT, Martial LR, Dossetor JB. The Canadian tax experience: A case study. The Non-Smokers' Rights Association (Canada) and The Smoking and Health Action Foundation (Canada). August, 1993.

Alaskans Voice Strong Support for Tobacco Tax Increase

Anne Marie Holen(1)

Almost three-quarters of Alaskans 18 and over support a major increase in state tobacco tax rates, according to a recent survey conducted by Mathematica Policy Research of Princeton, New Jersey. The survey was funded by the Robert Wood Johnson Foundation which supports tobacco control activities in Alaska through a SmokeLess States grant to the Alaska Native Health Board.

The survey was conducted to assess public support for a $1 per pack increase in the state cigarette tax. Such an increase has been specified in several bills introduced in the Alaska Legislature this session, and was included in the recommendations of the Long Range Financial Planning Commission as a way to raise new revenue and reduce future health care costs in the state by discouraging tobacco consumption.

Because initial publicity in Alaska about the tobacco tax proposal was largely in the context of addressing the state's fiscal gap, the survey included questions about that issue. Responses revealed that 74% of Alaskans have heard about the fiscal gap and that 72% believe that the fiscal gap can be closed only with a mix of spending cuts and new revenue sources.

The survey also provided side-by-side comparisons of public support for various measures to reduce the fiscal gap. From the responses, it is clear that alcohol and tobacco taxes are by far the most popular sources for new revenue. When asked to choose between alcohol and tobacco taxes, 31% indicated a preference for a tobacco tax, 29% for an alcohol tax, and 40% had no preference. When compared to other measures such as a motor fuels tax, state income tax, and state sales tax, preference for a tobacco tax increase ranged from 68% to 76%.

Other questions and answers include the following:

Now, I would like to turn to tobacco taxes. The current Alaska state tax is 29 cents per pack of cigarettes. In general, do you favor or oppose increasing Alaska's tobacco taxes by one dollar per pack to help close the state's fiscal gap?

	Tobacco Use				Political View		
	Non-users	Users	Both		Liberals	Moderates	Conservatives
Strongly Favor	26%	49%	60%	Strongly Favor	53%	52%	45%
Somewhat Favor	29%	25%	24%	Somewhat Favor	20%	23%	30%
Somewhat Oppose	9%	5%	4%	Somewhat Oppose	1%	8%	5%
Strongly Oppose	30%	16%	9%	Strongly Oppose	17%	15%	16%

If it was proven that a large cigarette price increase prevents or reduces smoking among children and teenagers, would you favor or oppose raising the state tax by one dollar per pack?

	Tobacco Use				Political View		
	Non-users	Users	Both		Liberals	Moderates	Conservatives
Strongly Favor	81%	53%	72%	Strongly Favor	74%	73%	72%
Somewhat Favor	12%	19%	14%	Somewhat Favor	10%	15%	14%
Somewhat Oppose	1%	7%	3%	Somewhat Oppose	2%	3%	2%
Strongly Oppose	6%	17%	9%	Strongly Oppose	8%	9%	10%

Do you generally favor taxing other tobacco products, such as chewing tobacco, snuff, and cigars, at a higher rate than cigarettes, about the same rate as cigarettes, or at a lower rate than cigarettes?

	Tobacco Use				Political View		
	Non-users	Users	Both		Liberals	Moderates	Conservatives
Higher Rate	8%	8%	8%	Higher Rate	5%	8%	5%
Same Rate	84%	79%	83%	Same Rate	81%	79%	89%
Lower Rate	4%	9%	6%	Lower Rate	9%	8%	4%

[After reading pro- and anti-tax arguments] *Now that you've heard this information, I want to get your final opinion, even if it is different from the opinions you expressed earlier. All things considered, do you favor or oppose increasing Alaska's tobacco taxes by one dollar per pack?*

	Tobacco Use				Political View		
	Non-users	Users	Both		Liberals	Moderates	Conservatives
Strongly Favor	70%	31%	58%	Strongly Favor	55%	60%	59%
Somewhat Favor	13%	20%	15%	Somewhat Favor	18%	15%	15%
Somewhat Oppose	5%	9%	7%	Somewhat Oppose	6%	6%	6%
Strongly Oppose	9%	37%	18%	Strongly Oppose	20%	17%	17%

CONCLUSION

The results of the Mathematica survey are consistent with data from other states which show strong public support for major increases in tobacco taxes. The Coalition on Smoking OR Health, comprised of the American Cancer Society, American Lung Association, and American Heart Association at the national level, has recommended an increase in federal cigarette excise taxes of $2 per pack, with an additional minimum $1 per pack cigarette tax imposed in each state. Currently, although Alaska has one of the highest rates of smoking and smoking related deaths in the country, the state cigarette tax is below the national average.If the tobacco tax legislation being considered by the Alaska Legislature is enacted into law, Alaska will become the first state to break the $1 per pack barrier for state cigarette taxes.

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(1) Alaska Native Health Board Trampling Tobacco Project, 1345 Rudokof Circle, Anchorage, AK 99508.

Criminal Deception

Ronald M. Davis, MD(1)

On November 10-12, 1995, the Northeastern University School of Law in Boston sponsored a conference entitled "Attorneys General Conference on Tobacco, Youth and the Public Health: Opportunities for Action." The purpose of the conference was to bring together Attorneys General (or their representatives) to discuss state lawsuits against tobacco companies seeking reimbursement for state costs from smoking-attributable disease (principally health care costs in the Medicaid program). At the time the states of Florida, Minnesota, Mississippi and West Virginia had launched such suits. Since then Massachusetts has joined the fray, and several other states are considering similar action. The conference was attended by Attorneys General or their representatives from the states involved in this litigation, as well those from a few dozen other states that were contemplating filing their own suits, or simply interested in gathering information.

I was asked to speak to the group on evidence that the tobacco industry markets its products to kids. Given the limited time available to address this broad topic, I decided to focus on evidence from the industry's marketing, advertising, and promotional activities. I did not review the internal industry documents that indicate the industry's interest in marketing their products to kids nor the scientific studies that demonstrate the impact of their promotions on youth. My presentation was organized along 17 different lines of evidence pointing in the direction of tobacco industry targeting of kids (see box).

CASH

At the outset it's important to consider the sheer volume of tobacco industry spending on cigarette marketing. In 1993 cigarette companies spent $6.03 billion to advertise and promote cigarettes, a twelve-fold increase over the $0.49 billion spent in 1979. When expenditures are adjusted for inflation, they still increased by 450% during this time period (1).

The expenditures in 1993 are equivalent to $190 *per second.* Imagine if that sum of money were made available to health advocates to sell health, instead of to the tobacco industry to sell death and disease.

This level of spending does not, by itself, speak to the issue of industry targeting of youth. But it is likely to have a huge impact on youth, by contributing to the ubiquity of cigarette advertising and promotion (see below).

Tobacco Industry Targets Youth

Cash
p**R**omotion
ub**I**quity
Marlboro miles . . .
Imagery
NIche marketing
Athletics
Language

pro**D**uct placement
v**E**nding machines
Copyright infringement
c**E**lebrities
Product giveaways
Toys
Instructions
vi**O**lations of TV ad ban
i**N**ternational

PROMOTIONS

Cigarette companies are shifting their marketing dollars from traditional forms of print advertising (on billboards, in magazines and newspapers, and at the point of sale) to promotional activities such as distribution of coupons and free samples, and sponsorship of sporting, cultural, and entertainment events. The proportion of the cigarette industry's expenditures on advertising and promotion that have been devoted to promotional activities has increased from 30% in 1980 to 84% in 1993 (1). Many of these promotions, such as free sample distribution and sponsorship of sporting events, reach children and teenagers (2-5), a fact that cannot be unknown to the industry.

UBIQUITY

As mentioned above, the cigarette industry spends a huge amount on advertising and promotion. With this largess cigarette companies are able to blanket our environment with images and messages that glamorize smoking. Tobacco billboards along our highways and in our communities are impossible for people — young and old — to ignore. Tobacco signs in stores (doors, clocks, change dishes, and so on), on grocery baskets, in trains and buses, at car races and tennis tournaments, in movies, on clothing, at video arcades, and on toys, are only a small portion of the pro-smoking cues to which we are regularly exposed.

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(1) Director, Center for Health Promotion and Disease Prevention, Henry Ford Health System, One Ford Place, 3C, Detroit, MI 48202-3450; and editor of the journal *Tobacco Control.*

The 1989 Surgeon General's report on smoking and health pointed out that "the ubiquity and familiarity of tobacco advertising and promotion may contribute to an environment in which tobacco use is perceived by users to be socially acceptable, or at least less socially objectionable and less hazardous than it is in fact."(6) Common sense dictates that this effect would apply to potential users (including children) as well as to current users.

MARLBORO MILES

A particularly pernicious type of promotion that has proliferated in recent years are cigarette continuity programs (or "frequent smoker" programs). These are promotional campaigns that provide free or discounted products (usually bearing tobacco brand names) to consumers for proof-of-purchase coupons or equivalent "currency" such as Marlboro Miles and Camel Cash. Recent evidence confirms the popularity of these campaigns among children and teenagers (7-10).

IMAGERY

The imagery in cigarette advertising makes it obvious that youth are a key target. Joe Camel describes smooth dating moves. The Marlboro man epitomizes the rugged individualism to which many young people aspire. An ad for Kool cigarettes shows a young, smug-looking couple — wearing jeans, tank tops, and high-top tennis shoes — standing next to a motorcycle, sending an unspoken message about rejecting authority (Figure 1). Sexual themes and imagery in cigarette ads heighten the raging hormones of our kids. Virginia Slims, Super Slims, Silva Thins, Capri, and other women's brands tout the thinness of the cigarette, a thinly veiled message to young girls and women about the weight-losing effect of smoking.

NICHE MARKETING

Tobacco companies use specific media to target niches in the marketplace, including youth. In the late 1960s the industry offered to voluntarily pull its advertising from television and radio because of its "substantial audience of young people" (11). Nevertheless, it advertises heavily in *Glamour*, one fourth of whose readers are girls under age 18, and in *Sports Illustrated*, one third of whose readers are boys under age 18. For a number of years R.J. Reynolds was the exclusive advertiser in a magazine called *Moviegoer*, which was distributed free in hundreds of movie theaters around the country; one half of those who attend movie theaters are less than 21 years of age (3).

ATHLETICS

The cigarette industry's voluntary advertising code includes a provision stating that "Cigarette advertising shall not ... show any smoker participating in, or obviously just having participated in, a physical activity requiring stamina or athletic conditioning beyond that of normal recreation" (12). However, a Kent ad shows a male tennis player, presumably having just finished his match, with a cigarette (Figure 2). A Vantage ad show a female ballet dancer, who appears to have just completed a vigorous workout, with a cigarette (Figure 3). Cigarettes are heavily advertised in newspaper sports sections, and at times the separation between editorial and advertising content becomes blurred (e.g., a Marlboro Sports Calendar, showing game schedules under a Marlboro banner). Sports associations lend their good name to cigarette promotions (e.g., Winston NBA sweepstakes, and NFL lighters provided free with the purchase of Winston cigarettes). Teenage sports enthusiasts are no doubt affected by these promotions.

LANGUAGE

Cigarette advertisers speak to youth in their own language. A simple two-letter word — YO — takes up one half of a full-page ad for Merit cigarettes. A tough-looking blond in a skin-tight suit, holding a cigarette, is "Totally Kool." Parliament ads promote the cigarette's "Perfect Recess." What do children think of, when they hear the word "recess"?

PRODUCT PLACEMENT

For years cigarette companies paid hefty sums of money for cigarettes and their brand names to be shown in movies. Perhaps the most well known example — and the most egregious — is the $40,000 spent by Philip Morris to place the Marlboro name throughout the kids' movie "Superman II" (13).

The manufacturer of Lark cigarettes reportedly paid $300,000 to place Lark images in the James Bond movie "License to Kill." After this arrangement became known and was criticized, the movie producer added a Surgeon General's health warning to the movie — shown at the end of the movie, after the credits.

Other examples, including several movies popular among kids, have been catalogued by Stop Teenage Addiction to Tobacco (STAT) (Springfield, MA).

VENDING MACHINES

Vending machines are another way in which children are encouraged to smoke. If we, as a society, were serious about enforcing the laws that exist in all 50 states prohibiting the sale of tobacco to minors, would we allow cigarette sales through vending machines? Would we ever consider the sale of alcoholic beverages through vending machines? If the tobacco industry were truly not interested in recruiting kids, wouldn't they support a ban on cigarette vending machines? Instead, we have a situation where most cigarette vending machines are unsupervised, uninspected, and unregulated. Some vending machines even sell cigarettes and candy out of the same machine!

The rules proposed by the Food and Drug Administration to curb minors' access to tobacco and their exposure to tobacco promotions would ban cigarette vending machines (14). The rules have been challenged in court by many tobacco and advertising interests.

COPYRIGHT INFRINGEMENT

Cigarette brand names are used on products targeted to kids. The best example is candy cigarettes, which come in several varieties (e.g., bubble gum, chocolate, or a hard sugary candy) (15). Candy cigarettes are often sold with brand names that are identical to those of real cigarettes, and with packaging and logos that are strikingly similar to those of their tobacco counterparts. In other cases, minor changes are made to the brand names (e.g., Cool instead of Kool, Lucky Spike instead of Lucky Strike, Pell Mell instead of Pall Mall, L&N instead of L&M). Research indicates that candy cigarettes may encourage smoking initiation among youth (16).

When the matter of candy cigarettes is raised, cigarette companies are always quick to point out that they don't manufacture these products. That may be true, but if these companies were not interested in kids, wouldn't they take action against this obvious copyright infringement?

Egregious examples are also seen in other countries with respect to American cigarettes. In Thailand, for instance, Winston kites and Marlboro school notebooks (Figures 4 and 5) have been distributed. In these cases, R.J. Reynolds and Philip Morris are guilty of one of two sins: 1) direct pandering to children, or 2) impotent action to prevent offensive copyright infringement.

CELEBRITIES

In their voluntary advertising code, the cigarette companies agreed to avoid the use of testimonials from athletes or other celebrities perceived to appeal to the young (5). But double standards abound. In Japan, the famous actor James Colburn appears in youth-oriented television commercials for Lark cigarettes. Closer to home, smokeless tobacco companies, which are not bound to the cigarette code, show no restraint in using athletes and other celebrities to hawk their products. Many famous athletes have been featured prominently in smokeless tobacco promotions, including Walt Garrison (football/Dallas Cowboys), Terry Bradshaw (football/Pittsburgh Steelers), George Brett (baseball/Kansas City Royals), Sparky Lyle (baseball/Texas Rangers), and Tom Seaver (baseball/Cincinnati Reds). In one ad, musician Charlie Daniels says, "When our band's cookin', the music smokes. But not me. Because like a lot of my friends I use smokeless tobacco." (17)

PRODUCT GIVEAWAYS

Non-tobacco products are often given away with the purchase of cigarettes. Many of these products have special appeal to children and adolescents, including sun glasses, cassette tapes, caps, a long cylindrical tube for cans of beer or soft drinks (with a shoulder strap for ease of carrying), and lighters with a picture of the helmet of the local professional football team (and the official logo of the National Football League).

From 1980 to 1993, cigarette industry expenditures for "specialty item distribution" increased from $69 million to $756 million. The proportion of total cigarette advertising and promotional expenditures devoted to these product giveaways increased during this period from 5.6% to 12.5% (1).

TOYS

Tobacco product brand names appear on toys and games. Philip Morris has distributed Marlboro frisbees at the gift shop at its Richmond, Virginia headquarters (Figure 6). Toy race cars emblazoned with the names of cigarettes (e.g., Marlboro) and smokeless tobacco products (e.g., Skoal, Copenhagen, Chattanooga Chew) have been sold in toy stores throughout the United State (Figure 7). Newport basketball games are seen in video arcades (18).

Overseas examples are evident too. In Buenos Aires, Argentina, on the occasion of the Eighth World Conference on Tobacco and Health in 1992, I picked up a toy Camel car (a photograph of which was published in Tobacco Control (19)). Three years later, at the next World Conference on Tobacco and Health, in Paris, I purchased a similar product. Also in Paris, I came across a Formula One children's ride, in which different "kiddie cars" bore stickers for Marlboro, Winston, and Camel (20).

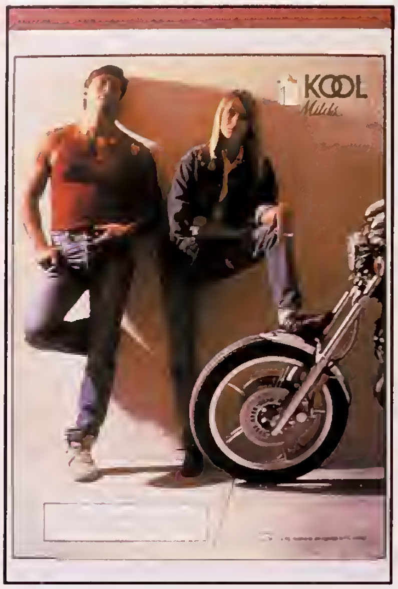

Figure 1. A Kool ad — targeting senior citizens?

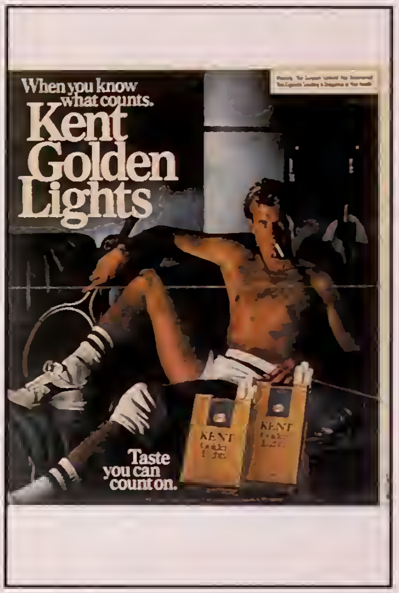

Figure 2. Kent tennis player — is this "normal recreation"?

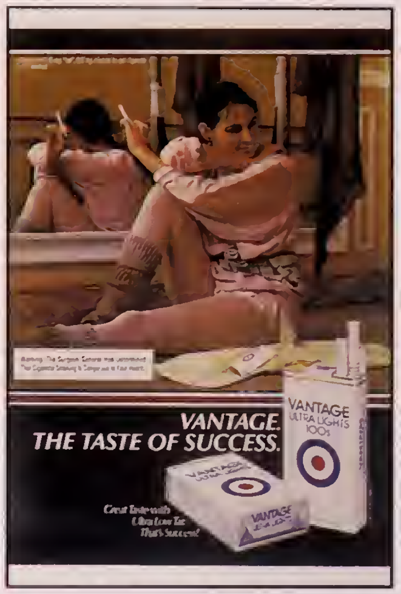
VANTAGE.
THE TASTE OF SUCCESS.
VANTAGE ULTRA LIGHTS 100s
Great Taste with Ultra Low Tar. That's Success!

Figure 3. Vanage ballet dancer — is this "normal recreation"?

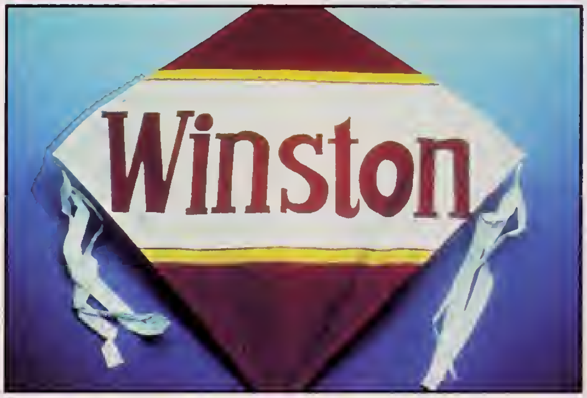

Figure 4. A Winston kite from Thailand

In some cases these promotions occur by direct action of the cigarette company (e.g., the Marlboro frisbee). In other cases cigarette company involvement may be more indirect, but again, these companies could prevent the use of their brand names in children's products through licensing arrangements or legal action, if they were truly interested in avoiding the youth market.

INSTRUCTIONS

Some smokeless tobacco advertising provides instructions on how to use the product. This clearly indicates that at least some advertising is aimed at recruiting new users, contradicting the "party-line" statements by tobacco and advertising industry spokespersons that the only effects of tobacco advertising are to promote brand loyalty and brand switching.

Former Dallas Cowboys star Walt Garrison, for example, "answers your questions about smokeless tobacco" in *Parade* magazine (June 8, 1980) and in other publications (21). One answer to a question states, "At first you could feel a slight irritation on the gum, and the tobacco may move around your mouth more than it should, and you might work up too much saliva. But learning is part of the fun, and these things pass with practice. Two weeks should make you a 'pro.'"

Similarly, a brochure distributed at the point of sale, with the headline "It's as easy as 1-2-3", gave instructions on how to use Skoal Bandits (21).

VIOLATIONS OF TV AD BAN

Cigarette advertising on television and radio was banned in 1971. Smokeless tobacco advertising in the broadcast media was banned in 1986. Nevertheless, these products continue to be promoted on television in a variety of ways. Tobacco sponsorship of athletic events, such as Marlboro car races or Virginia Slims tennis tournaments, leads to repeated mentions of the

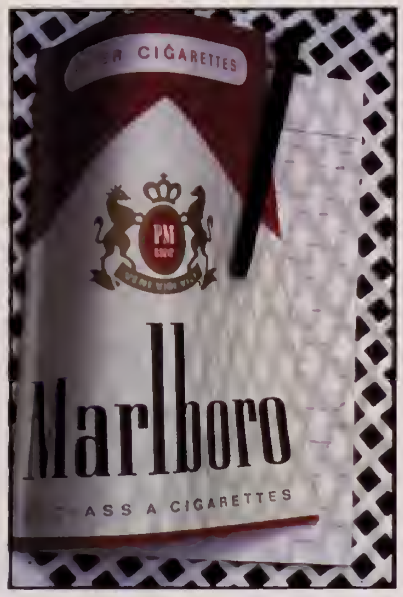

Figure 5. A Marlboro school notebook from Thailand.

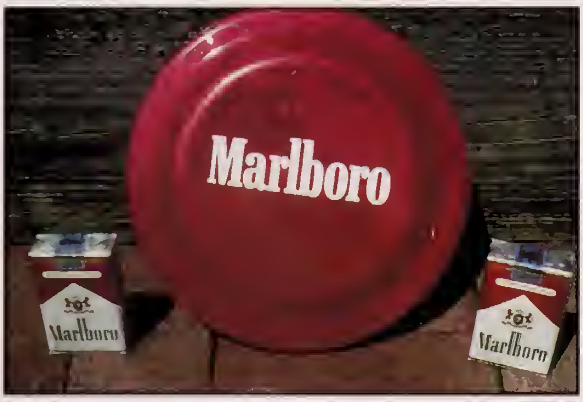

Figure 6. A Marlboro frisbee sold by Philip Morris.

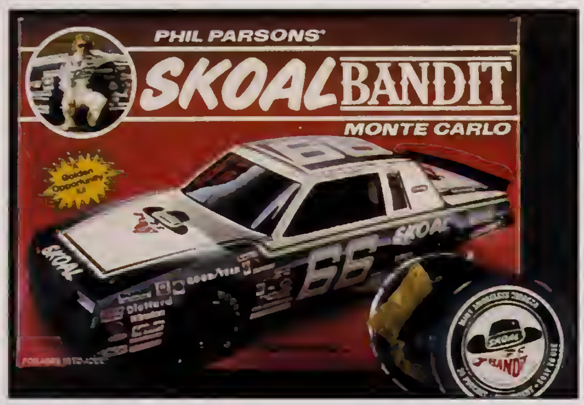

Figure 7. Skoal Bandit toy car "For ages 10 to adult."

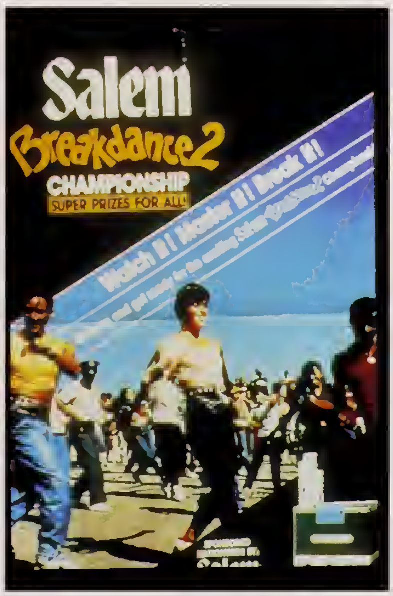

Figure 8. Salem sponsors a breakdance contest in Malaysia.

brand name by announcers and graphic displays of the brand name on the television screen throughout the broadcast. Signs and billboards at the events are captured by television cameras, and, in fact, these signs are often placed at strategic locations to maximize the likelihood that they will be picked up by cameras. Patches bearing the brand name appear on athletes' clothing, which often find their way onto television during the athletic competition and post-competition interviews. On February 23, 1995, the ABC program "Day One" aired a segment showing how sponsors of NASCAR car races train their racers (and presumably others who wear their emblems, such as pit crews) to slip the sponsor's name into a sound bite, and their emblems into camera shots, during media interviews (22). In one 94-minute broadcast of the 1989 Marlboro Grand Prix, the Marlboro name was seen or mentioned 5,933 times, and the name was seen for a total of 46.2 minutes, or 49% of the total broadcast time (4). How can the tobacco industry do this and at the same time profess to have volunteered to withdraw cigarette advertising from television and radio because of its "substantial audience of young people" (11)?

Last year the Justice Department, after two and a half decades of ignoring violations of the ban on broadcast cigarette advertising, began to take action against Philip Morris for placing cigarette signs near scoreboards and in other locations where they were likely to be captured by television cameras. However, this action does not extend to some sports (e.g., car racing) where tobacco sponsorship is dominant, and does not yet involve other cigarette companies.

INTERNATIONAL

Examples are given above where U.S. tobacco companies market their products overseas so as to appeal to, and lure in, very young customers. Other examples include advertisements promoting free tickets to a rock concert in Taiwan for returning five empty packs of Winston, Salem sponsorship of a break dance contest in Malaysia (Figure 8), and Salem sponsorship of television concerts in Hong Kong by U.S. pop stars Madonna and Paula Abdul (23).

CONCLUSION

The evidence that tobacco companies are targeting kids — presented only in abbreviated form here — is abundant. The industry's advertising and promotional campaigns demonstrate a callous, reckless, immoral disregard for the health and welfare of our children. When one considers evidence beyond marketing practices — from internal industry documents, patent applications, testimonials from researchers formerly employed by the industry, and so on (14) — a pattern emerges that can be summed up by two words: criminal deception.

The manufacture, promotion, sale, and use of tobacco products need to be regulated and controlled in a manner proportionate to the enormity of the damage they wreak on public health, the economy, the environment, and society. And the manufacturers and their co-conspirators — and the individuals who run those companies — must be held liable for the harm they cause.

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Tobacco Control and the American Medical Association

Thomas P. Houston, MD(1)

More than 400,000 Americans die prematurely each year from the combined effects of active smoking and exposure to environmental tobacco smoke (ETS). Smoking is the major cause of lung cancer, of deaths from emphysema and chronic bronchitis, and is a principal cause of heart disease and stroke. About one-half of smokers die from a tobacco-related illness or condition - almost 25% of the total deaths in the U.S. (1).

The decision to assume the enormous risks of smoking is not usually one made by informed adults. Nearly 90% of new smokers are under the legal age for purchase of tobacco products. Three thousand children begin regular smoking daily. Children discount the regular risks of smoking, overestimate their ability to quit, and believe that smoking is much more the norm among adults and their peers than is really the case.

Since 1847, the mission of the American Medical Association (AMA) has been "to promote the science and art of medicine and the betterment of public health." Former Surgeon General Dr. C. Everett Koop has described tobacco use as "the nation's number one public health problem." In the context of public health, tobacco use prevention and control has become an increasingly important part of the AMA's strategy.

The first mention of tobacco in the "Digest of Official Actions of the AMA House of Delegates" comes in a 1960 resolution for the AMA to "clarify its position regarding the harmful effects of tobacco ... and take a lead position in an educational campaign aimed at the youth of the United States." In the early 1980's, the volume of resolutions increased markedly. The actions called for were much more pointed and action oriented. Since then, the AMA has begun to act in ways that promote tobacco control principles, such as supporting a $2 increase in the federal excise tax on cigarettes. Prevention and control of tobacco use is one of the key issues in the American Medical Association's 1994-1996 strategic corporate plan.

In general, four key areas exist for policy development in tobacco control: curbing youth access to tobacco, protecting the public from the hazards imposed by ETS, restricting tobacco industry advertising and promotion, and encouraging higher excise taxes on tobacco products.

YOUTH ACCESS

The National Cancer Institute estimates that at current teen smoking rates, five million American children will die prematurely from having begun to smoke. In many states communities have enacted laws that would keep children from purchasing tobacco. The key tobacco industry strategy is to introduce weak legislation at the state level which expressly preempts communities from passing their own effective laws or ordinances. Specifically the industry opposes effective legal barriers such as licensing tobacco vendors and giving local authorities enforcement power.

The industry prefers: legislation which provides fines only for those who "knowingly" sell tobacco to children; outlawing public health research and investigative reporting about illegal sales; holding the child purchaser not the merchant-seller responsible for the illegal sale. Lobby and state testimony by medical society members should be focused on meaningful legislation that will actually prevent tobacco sales to children.

ENVIRONMENTAL TOBACCO SMOKE

In 1993 environmental tobacco smoke (ETS) was officially classified as a Class A carcinogen by the Environmental Protection Agency. ETS causes 50,000 premature deaths annually. The serious public health threat of ETS demands attention from the health community as well as government agencies. Since laws that restrict smoking in business and public places reduce consumption, the tobacco industry will fight them to increase its profits. Health providers and medical societies should actively support clean indoor air regulations and legislation.

PROMOTION

The tobacco industry spends over $5 BILLION yearly to promote its products! Recently much of this has been directed at youth. Overall, 85% of youth purchases are focused on the most highly advertised brands in the US — Marlboro, Camel, Newport. Promotion of the race car circuit, clothing and trinkets with brand logos, and distribution of free samples have increased dramatically in recent years. Monitoring youth activities targeted by the industry and countering them with pro-health messages can be useful strategies for organized medicine.

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(1) Director, Department of Preventive Medicine & Public Health, American Medical Association, 515 N. State St, Chicago, IL 60610.

TAXATION

Since the landmark Surgeon General's report in 1964, taxes on tobacco, adjusted for inflation, have *dropped* about 28%. The percentage of the retail price of tobacco attributed to taxes has dropped from 50% to 25%. Since 1964, tobacco industry profits have risen considerably because of frequent, substantial raises in the wholesale prices of cigarettes. In recent years, tobacco price increases have risen at about three to four times the rate of inflation, or about 12% per year.

Tobacco pricing makes a difference in consumption and can be a potent force in saving lives, particularly among young people. A 10% increase in price leads to about a 4% drop in smoking (2). The AMA and other voluntary health agencies estimate that a $1 increase in the federal excise tax on tobacco would eventually save one million lives. The AMA strongly advocates increased taxes on tobacco products as a way to reduce the number of teen smokers.

The AMA recognizes that success in tobacco control is more likely to occur at the state and local level, especially considering the powerful influence of the tobacco industry in Washington, DC. The AMA's House of Delegates not only accepts policy recommendations from state medical societies, but has passed several statements calling for action on their part. One such recommendation encourages state societies to "attempt to raise the state excise tax on tobacco products (490, 948)."

In some states, such as North Carolina and Texas, medical societies have created tobacco control subcommittees as an official part of the society structure to propose policy and develop action plans designed to impact tobacco use (3). In coordination with the lobbying and educational activities traditionally taken on by most state medical societies, such efforts can be very productive in targeting tobacco control issues such as tobacco pricing.

Involvement with other groups such as the local chapters of the American Cancer Society, American Heart Association, American Lung Association, Hospital Association, Dental Society, and state chapters of medical specialty societies is also very effective. Alaska has a very active statewide tobacco control coalition, Alaska Tobacco Control Alliance (ATCA). Active participation of the medical society at both state and local levels is needed.

Finally, a word about action and activism. For too long, organized medicine has been accused of armchair activism — of only being involved in traditional activities that are safe, comfortable, and avoid risk. It is too little, too late to be content with generic smoking cessation advice to our patients or sponsoring a health booth at the county fair. We must take our message outside the walls of the office and hospital, to confront the industry and its apologists directly (4,5). Testimony from the health community at city council hearings on the benefits of a tobacco tax increase can refute the propaganda and scare tactics of the tobacco industry. Medical societies can make a tobacco tax increase a special priority, and shepherd an ordinance through the system. The media is a very useful way to reach the public - most newspapers welcome editorials and letters on tobacco control issues from physicians and medical societies. The potential to make a difference is present if we take the time to get involved. Together, the AMA and its partners in the states have an opportunity to make a difference, working to ease the human and economic toll taken by tobacco.

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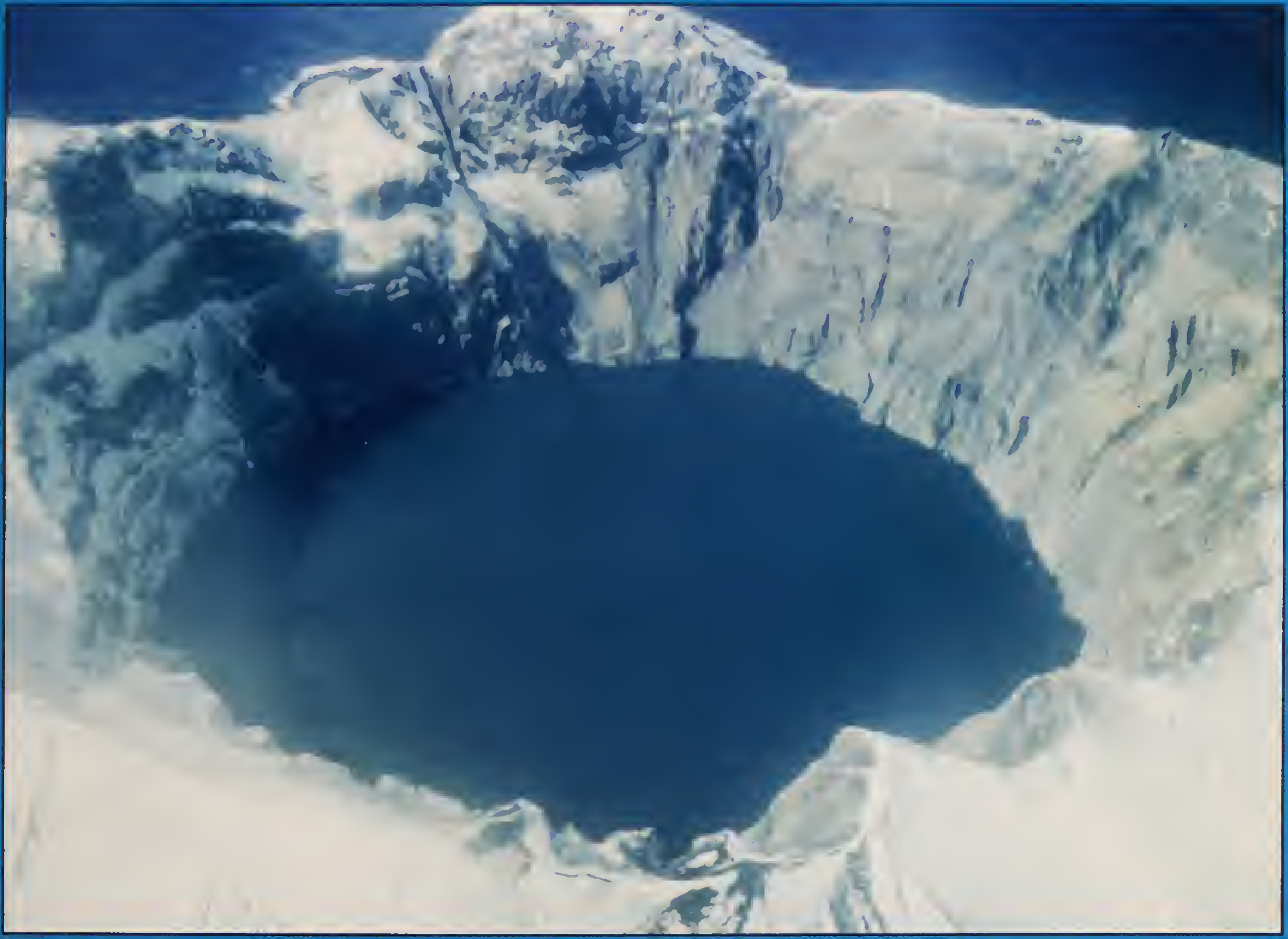

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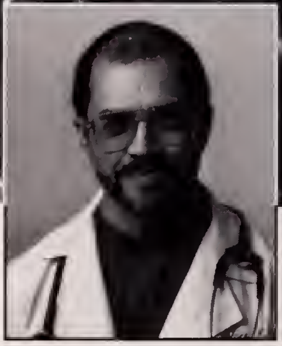

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About the cover: Kasatochi Island, Aleutian Islands. Kasatochi is the summit of a volcano whose crater is water filled. Since the surface of the water is approximately 130 feet above sea level (satellite observations), the water is almost certainly fresh. Kasatochi is located just north of the west end of Atka. Photo courtesy of Donald R. Rogers, M.D., Anchorage, Alaska.

A Population-Based Study of Paralytic Shell Fish Poisoning in Alaska

Bradford D. Gessner, MD(1)
Mindy Schloss, MPH(1)

ABSTRACT

During May and June 1994, the authors interviewed and requested a shellfish sample from a population-based sample of 170 residents from Kodiak and Old Harbor, Alaska. Of 51 Old Harbor and 68 Kodiak residents who had eaten shellfish gathered from Kodiak Island, 18 and 6 percent, respectively, had a history of PSP. We calculated the incidence of paralytic shellfish poisoning in Old Harbor and Kodiak as 15 and 1.5 per 1000 persons per year, respectively. Of 12 butter clam batches collected from residents, 6 had a paralytic shellfish poison toxin level greater than the regulatory limit of 80 µg saxitoxin equivalent per 100 g of tissue; one of the 29 people who ate these shellfish developed illness. People who eat shellfish collected from non-commercial beaches have a high rate of paralytic shellfish poisoning. It may be possible to raise the regulatory level for paralytic shellfish poison toxin without affecting the public health.

Paralytic shellfish poisoning (PSP) results from the ingestion of shellfish contaminated with one or more components of a group of toxins including saxitoxin, neosaxitoxin, and gonyautoxin (1). Paresthesias, gastrointestinal symptoms, respiratory arrest, and death may occur (2,3). Outbreaks of PSP have been reported worldwide and some evidence exists that the incidence of PSP is increasing (4).

Many Alaskan and other U.S. coastal residents harvest shellfish for subsistence or recreational use. By doing so they place themselves at an unknown risk of PSP since the population-based incidence at various locations is unknown and, due to a variety of logistic and financial constraints, monitoring of shellfish toxin levels at non-commercial beaches seldom occurs. Even where monitoring occurs, the optimum regulatory toxin level is not known and residents may ignore beach closure announcements. No information exists on the beliefs and knowledge which allow people to continue to harvest shellfish despite this unknown risk of illness.

Alaska has one of the highest incidences of reported PSP in the world and a large population who frequently eat shellfish collected from unmonitored non-commercial beaches; for example, residents in Old Harbor, AK collected an average of eight kilograms of butter clam tissue per person during 1991 (Alaska Department of Fish and Game, Division of Subsistence). We conducted the current investigation to estimate the risk of PSP among recreational and subsistence shellfish harvesters and to develop rational recommendations for shellfish harvesting from non-commercial beaches. To this end, we estimated the number of persons with a previous history of PSP, determined the characteristics and knowledge of people who harvest shellfish for subsistence or recreational use, and determined the paralytic shellfish poison level in shellfish which are eaten in non-outbreak settings.

MATERIALS AND METHODS

Background

Our investigation was prompted by the occurrence during May and June 1994 of five outbreaks of PSP involving 12 persons - including four who required mechanical ventilation - in the towns of Kodiak (1994 pop. 7,428 plus 6,818 along the Kodiak road system) and Old Harbor (1994 pop. 311) on Kodiak Island, AK. We did not include persons involved with these outbreaks, or their family members, in the present investigation.

Epidemiologic investigation

To identify subjects in Kodiak, we randomly selected 108 private numbers from the Kodiak telephone directory and interviewed household residents by telephone. In Old Harbor, we went to each house in the village since telephones are much less common than in

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(1) Alaska Division of Public Health, 1231 Gambell St, Anchorage, Alaska, 99501; tel. (907)-278-2990; fax. (907)-274-1384

Kodiak; residents were located in 22 households. In both locations we attempted to interview all persons living in the household at least 15 years of age. For each person we collected information on basic demographics, years of residence on Kodiak Island, previous shellfish harvesting on Kodiak Island, knowledge regarding PSP, and beliefs regarding methods of preventing PSP. Not all persons provided all of the information we requested. Thus, for some variables data is incomplete.

PSP incidence

We asked each person whether they had ever had numbness or tingling of the lips or hands within 6 hours of eating shellfish; we did not ask them to specify a date of illness occurrence because of concerns regarding recall bias. From this information we determined two quantities: the population-based PSP incidence rate per 1000 persons per year and the rate of persons with illness per 1000 persons per year. These were calculated as follows:

$$\mathbf{I_{I/P} = (N_{I/P} \div Y) \infty P \infty 1000}$$

Where:

$I_{I/P}$ =	The PSP incidence rate or persons with illness per 1000 persons per year.
$N_{I/P}$ =	The number of illness episodes or persons with illness.
Y =	The sum of years of shellfish harvesting for all persons interviewed.
P =	The proportion of the population who gather shellfish each year.

Analysis of risk factors for PSP

We determined whether any of the following risk factors were associated with a previous history of PSP: gender, city of residence, age, race, first language, years of residence on Kodiak Island, knowledge that eating shellfish could cause PSP, and knowledge that PSP could cause death. Risk ratios for illness and their 95% confidence intervals were calculated using Epi-Info computer software, version 6.01 (5).

Surveillance data-base PSP incidence

To compare the PSP incidence measured during our investigation with the PSP incidence reported through passive surveillance, we reviewed a reportable disease surveillance database maintained by the Alaska Division of Public Health since 1973. We identified residents of Old Harbor or Kodiak with PSP who had been detected by this surveillance system and used this number to estimate the PSP incidence.

Shellfish toxin levels

To determine paralytic shellfish poison toxin levels in shellfish which were routinely eaten by island residents, we asked each person we interviewed whether they had shellfish in their homes which they had collected during 1994 from a beach on Kodiak Island and, if they did, we asked for a sample. Only one person from whom we requested a sample declined. For each resident from whom we collected a sample we attempted to determine the number of persons in the household who had eaten shellfish from the tested batch and the quantity of shellfish from the tested batch which had previously been eaten.

Paralytic shellfish poison toxin concentrations in shellfish obtained from residents were determined by the U.S. Food and Drug Administration. Laboratory analyses were performed using the standard mouse bioassay (6). Standardized tables were used to convert paralytic shellfish poison concentration from mouse units to µg saxitoxin equivalent per 100 g of tissue (7).

RESULTS

Descriptive characteristics

We collected information for 117 Kodiak and 53 Old Harbor residents. Overall, 54% were female; 50% were Caucasian, 39% Alaska Native, 9% Filipino, and 2% other races; and age ranged from 15 to 80 years (median, 37). English was the first language for 79% and among the remainder, 11 different first languages were identified.

Old Harbor and Kodiak residents had similar knowledge that eating shellfish can cause PSP and that PSP can cause death (Table 1). Other characteristics varied

Table 1.
Characteristics of persons interviewed about paralytic shellfish poisoning (PSP); Kodiak and Old Harbor, Alaska; May, 1994.

Characteristic	Kodiak	Old Harbor
Number interviewed	117	53
Alaska Native	10%	89%
Knowledge that eating shellfish can cause PSP	85%	94%
Knowledge that PSP can cause death	74%	79%
Harvested shellfish during 1994	28%	75%
Previous consumption of shellfish harvested from Kodiak Island	58%	96%
Mean years of residence (range)	14 (0, 60)	32 (0, 72)
Mean years of shellfish harvesting among shellfish harvesters (range)	13.7 (<1, 50)	31.1(<1,72)

considerably between the two villages. Compared to Kodiak residents, Old Harbor residents had resided in their town for more than twice as many years, previously eaten shellfish from Kodiak Island almost twice as frequently, harvested shellfish for over twice as many years, and harvested shellfish during 1994 twice as frequently. Among persons who had eaten shellfish harvested from Kodiak Island, 77 percent had done so their entire lives.

The most common methods mentioned for preventing PSP were eating shellfish only during the winter (28 percent), not eating shellfish when a red tide was present (23 percent), eating shellfish only during months with the letter "r" in the spelling (14 percent), cooking the shellfish (12 percent), eviscerating the shellfish (6 percent), chewing the shellfish first and observing for peri-oral paresthesias (4 percent), not eating shellfish which when cooked with a silver coin turned the coin black (3 percent), and allowing a pet to sample the shellfish first (2 percent). Thirty-six percent of people said they did not know a reliable method for preventing PSP.

Table 2.
Determination of paralytic shellfish poisoning (PSP) incidence among persons who reported previous consumption of shellfish collected from Kodiak Island; Kodiak and Old Harbor, AK; May, 1994.

Measurement	Kodiak	Old Harbor
Number of persons with a previous history of PSP	4 (5.9%)	9 (18%)
Persons with PSP (per 1000 exposed population* / yr.)	4.3	5.6
Persons with PSP (per 1000 total population / yr.)	1.2	4.2
Number of previous PSP incidences	5	25
PSP incidence (per 1000 exposed population* / yr.)	5.4	15.7
PSP incidence (per 1000 total population / yr.)	1.5	15.0

*Those persons who reported eating shellfish collected from Kodiak Island during at least one year.

PSP incidence

Among 68 Kodiak and 51 Old Harbor residents who had previously eaten shellfish harvested from Kodiak Island we identified four (6 percent) and nine (18 percent), respectively, who had experienced numbness or tingling of the face or hands within 6 hours of eating shellfish (Table 2). While the number of persons with PSP per total population differed between Kodiak and Old Harbor, the number of persons with PSP per total exposed population (i.e., those who had previously eaten shellfish collected from Kodiak Island) was similar in the two towns.

In Kodiak, one person experienced two episodes of PSP for a total of five episodes, while in Old Harbor one person experienced two episodes and one each had experienced eight and nine episodes for a total of 25 episodes. All four persons with multiple episodes of PSP were Alaska Native and reported eating shellfish collected from Kodiak Island for from 42 to 49 years. With the addition of 17 cases occurring in two Old Harbor residents, the total population and exposed population based incidence rates were considerably higher in Old Harbor than Kodiak (Table 2).

Among the 13 people from either location with previous symptoms of PSP, only one stopped eating shellfish as a result of their illness. While we did not estimate the year in which most previous episodes had occurred, we did note that two had taken place during 1994, on May 24 and 25.

Analysis of risk factors

Among people who previously had gathered shellfish from Kodiak Island, we identified four risk factors for a previous history of PSP including years of residence on Kodiak Island, Alaska Native race, years eating shellfish collected from Kodiak Island, and residence in Old Harbor (Table 3). Knowledge that eating shellfish causes PSP and that PSP can cause death were not associated with a history of PSP.

Surveillance data-base PSP incidence

During 21 years of reporting, the surveillance database identified 11 persons from Old Harbor and 17 persons from Kodiak who had been reported with PSP. Based on 1994 population data, this implies a reported incidence rate of 1.7 and 0.06 cases per 1000 persons per year in Old Harbor and Kodiak, respectively.

Shellfish toxin levels

We tested 12 batches of butter clams obtained from 10 households which had been collected on Kodiak Island for subsistence use by residents during February to May 1994. All samples had been collected before the outbreaks which prompted this investigation. An estimated 49 people had eaten shellfish from one of these batches. Paralytic shellfish poison levels ranged from 38 to 330 μg saxitoxin equivalent per 100 g of tissue (mean,

Table 3.

Risk factors for a history of paralytic shellfish poisoning (PSP); Kodiak and Old Harbor, AK; May, 1994.

Risk factor	Proportion with a history of PSP	Risk ratio (95% CI*)
Years living on Kodiak Island		
≥ 23 (N=60)	0.20	11.8 (1.6, 87.9)
<23 (N=59)	0.017	
Race		
Alaska Native (N=60)	0.20	11.6 (1.6, 86.4)
Non-Native (N=58)	0.017	
Years eating shellfish from Kodiak Island		
≥ 20 (N=60)	0.18	5.4 (1.3, 23.4)
<20 (N=59)	0.033	
Residence		
Old Harbor (N=51)	0.18	3.0 (1.0, 9.2)
Kodiak (N=68)	0.056	
Age		
≥ 40 years (N=60)	0.15	2.2 (0.7, 6.7)
<40 years (N=59)	0.068	
Gender		
Female (N=58)	0.14	1.7 (0.6, 4.9)
Male (N=61)	0.082	
Knowledge that PSP can cause death		
Yes (N=100)	0.11	1.0 (0.3, 4.3)
No (N=19)	0.11	
Knowledge that eating shellfish causes PSP		
Yes (N=110)	0.12	Undefined
No (N=9)	0	

*Confidence interval

146). Six batches, from six different households and from which 29 people had eaten, had PSP toxin levels greater than the regulatory limit of 80 µg saxitoxin equivalent per 100 g of tissue; three batches had shellfish toxin levels >200 µg saxitoxin equivalent per 100 g of tissue. One 58 year old female from Old Harbor recalled possibly having had peri-oral paresthesias after eating shellfish; when tested, her leftover shellfish contained 118 µg saxitoxin equivalent per 100 g of tissue. No symptoms were noted by any of the other persons questioned. People were unable to recall the quantities of shellfish eaten, but persons from all households had eaten from the tested batch of shellfish on multiple occasions.

DISCUSSION

We documented PSP incidence rates of 15.0 and 1.5 cases of PSP per 1000 population per year in Old Harbor and Kodiak, respectively; we have likely underestimated PSP incidence since mild cases of PSP may not have been recalled. If our results are reproducible in other communities, it suggests a substantial risk of illness among persons who eat shellfish from uncertified beaches. By comparison, the reported U.S. incidence of salmonellosis, one of the most common food-borne illnesses, was 0.2 per 1000 population during 1991 (8).

Compared to active case-finding, the estimated incidence from the reportable disease database was 10 to 30-fold less, although during active case-finding we interviewed persons at least 15 years of age while the surveillance system incidence estimates were determined for all ages. Because people with serious symptoms of PSP are likely to report to health care facilities, we do not believe the surveillance database failed to detect many serious cases of illness. Our data, however, indicate that substantial underascertainment of mild cases occurs. It is particularly disturbing that we identified two cases of illness which occurred shortly before the five known outbreaks. Because the occurrence of dinoflagellate blooms which result in paralytic shellfish poison production cannot be predicted (6,9), mild illness should be considered a sentinel event. Public health officials should educate communities to report immediately all cases of PSP to a health care provider.

Analysis of risk factors found that Alaska Natives, Old Harbor residents, and people residing on and collecting shellfish from Kodiak Island for the longest period had the highest risk. Paralytic shellfish poison composition and concentration within shellfish species may change by location, depending on local dinoflagellate populations, currents, tidal flows, and ocean salinity (1,4). Theoretically, it is also possible that susceptibility to paralytic shellfish poison toxins differs between people based on immune status (10,11) or sodium channel structure differences (12). In this instance, however, we believe the most likely explanation is that the persons most likely to develop illness, and thus the people to whom prevention efforts should be directed, are those persons most frequently exposed, predominantly Alaska Natives and rural residents who depend upon shellfish harvesting for maintenance of a subsistence life-style.

A comparison of PSP incidence rates in Old Harbor and Kodiak further supports this conclusion. The tenfold difference in incidence rate likely represents a combination of the effects of two outliers who accounted for 68 percent of reported PSP cases in Old Harbor and the difference in exposure to shellfish. If the number of persons with a previous history of PSP rather than the number of PSP cases is used as the numerator and persons with a history of eating shellfish collected from Kodiak Island rather than the total population is used as the denominator, the rates are similar for both towns.

The Alaska Division of Public Health and the Alaska Department of Environmental Conservation recommend that shellfish collected from untested beaches in Alaska should never be eaten. Our investigation showed, however, that 96% of Old Harbor and 48% of Kodiak residents had previously harvested shellfish, some for over 70 years, and many of these persons had harvested shellfish during 1994. Residents ate shellfish despite awareness that PSP resulted from eating shellfish and that PSP could cause death. This may be because almost two-thirds of residents believed that it was possible to collect, prepare, or test shellfish in such a way that PSP could be prevented.

The most prevalent belief, stated by 42% of persons, was that harvesting outside of late spring and early summer would prevent PSP. A previous report from Alaska, however, demonstrates that although the majority of outbreaks occur during May and June, outbreaks of PSP occur during all seasons (13). Others have reported that some species of shellfish, particularly butter clams, can retain poison for up to 2 years following exposure (2,14), suggesting that even if dinoflagellate blooms are seasonal, shellfish may be toxic year round. Another 35% of people stated that not eating shellfish when a "red tide" was present or cooking the shellfish would prevent PSP. Previous research, however, has suggested that cooked shellfish may retain toxicity (13). Additionally, not all toxic dinoflagellate blooms will appear red (1,4); for example, among the five outbreaks that prompted this investigation, no red tide had been observed before the occurrence of illness. Finally, 6% of persons believed eviscerating the shellfish would prevent PSP. Again, data from Alaska suggest that in some instances enough toxin is retained in the shellfish tissue after evisceration to cause illness (13).

Because most people knew of the dangers presented by PSP and still harvested shellfish, future education efforts might be most successful if they focus more on beliefs about prevention methods and less on the origin and outcome of PSP. In addition, the long history of subsistence and recreational shellfish harvesting among coastal residents, and the dependence of many coastal residents on a subsistence life-style, suggests that prevention efforts aimed at eliminating shellfish harvesting may not be successful. Unfortunately, little data exists on methods of decreasing risk. In the absence of an inexpensive, accurate field test for paralytic shellfish poison toxin, future investigations should evaluate temporal, geographic, and species toxin level variations. The goal of these investigations should be the development of recommendations for decreasing, but not necessarily eliminating, risk associated with eating shellfish collected from non-commercial beaches.

In areas where monitoring of non-commercial beaches exists, accurate determination and reporting of toxin levels dangerous to human health will help to further decrease risk. While previous reports have documented illness after exposure to shellfish with low toxin concentrations (13), we found that residents routinely eat shellfish with toxin levels as high as 330 μg per 100 g of tissue without developing overt symptoms. Our data suggest that it may be possible to raise the current regulatory limit of 80 μg saxitoxin equivalent per 100 g of shellfish tissue without seriously affecting the public health, although larger studies are needed to confirm our results. To establish credibility and thus to influence behavior, recommendations to open or close non-commercial beaches must accurately reflect the danger to the individual consumer. The public may respond more consistently to closing beaches infrequently and only when significant risk exists than to frequent closures when risk is minimal.

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Tribal-based Cancer Control Activities Among Alaska Natives: Services and Perceptions

Arthur M. Michalek, Ph.D.(1)
Martin C. Mahoney, M.D., Ph.D.(2)
Mia Papas, B.S.(3)
Martha Tenney, M.P.H.(4)
Linda Burhansstipanov, M.S.P.H., Dr.P.H.(5)

ABSTRACT

Cancer has become a significant health problem in American Indian and Alaskan Native (AI/AN) communities. Despite the precipitous rise in cancer rates, limited data are available concerning cancer control services operative in these communities. To address this issue, a cross-sectional survey of all federally recognized tribes was undertaken to ascertain the breadth of cancer control activities offered and Tribal Health Directors perceptions of and priorities ascribed to cancer. This article presents responses given by AN Health Directors juxtaposed to those proffered by AI Health Directors. Nearly three-quarters (71%) of respondents perceived cancer rates to be increasing. Cancer was found to rank third, fifth among AI Health Directors, among seven health conditions when Directors were asked to rank their Tribe's commitment to confronting each one. Awareness of cancer as a public health concern coupled with competing health problems relegates cancer control activities to a lower priority. Findings underscore the need to elevate the issue of cancer in Indian Country as well as to educate investigators to become more sensitive and responsive to other Tribal health issues.

INTRODUCTION

Cancer ranks as a leading cause of death among Native Americans (the third and second leading cause of death among males and females, respectively). Among Alaska Natives, cancer ranks as the number one cause of death among females and the second leading cause of death among males (1). Despite these statistics little effort has been directed toward identifying the extent of cancer control services available to Native Americans (Alaska Natives and American Indians).

We previously conducted a survey of State Health Departments to assess the extent of cancer control programs targeted to Native Americans (2). As a sequel to the State Health Department Survey, a survey of American Indian/Alaska Native (AI/AN) Tribal Health Directors was undertaken to determine their perceptions about cancer and the extent of cancer control programming directed by the Tribe (3). Germane to this article are results obtained from Alaska Native (AN) Health Directors. Their responses will be reported and juxtaposed with those obtained from American Indian (AI) Health Directors from the lower 48 to provide a more comprehensive illustration of available health services and unmet needs.

METHODS

The primary objective of the original survey was to document the extent of cancer prevention and control programs provided by the Tribes; and to document Tribal Health Directors' perceptions of and priorities ascribed to cancer. The specific aims of the study were to document the availability and extent of cancer control programs, identify Tribal Health Director perceptions of cancer patterns, ascertain priorities ascribed to cancer relative to other health problems and identify Tribally developed cancer control materials.

This study employed a cross sectional survey of Tribal Health Directors from all federally recognized

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(1) Assistant Director for Educational Affairs, Roswell Park Cancer Institute.

(2) Assistant Professor, Roswell Park Graduate Division & Department of Family Medicine, School of Medicine & Biomedical Sciences, SUNY at Buffalo.

(3) University of Massachusetts at Amherst.

(4) Public Health Educator, Native American Cancer Research Program, AMC Cancer Research Center.

(5) Director, Native American Cancer Research Program, AMC Cancer Research Center .

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AI/AN tribes (N=535). Two mailings with a sample of telephone reminders resulted in an approximate 30% response rate. A significantly lower proportion of AN Health Directors responded (19%; n=42) than did AI Health Directors from the lower 48 (34%; n=108). A sample of non-respondents were subsequently contacted by telephone to determine reasons for not responding. Many felt that the survey should best be filled out by the area IHS office; others were involved in issues related to compacting; and others, especially in Alaska, were involved in sustenance (fishing season).

The survey solicited responses to a variety of questions including respondent and Tribal characteristics; perceptions of temporal trends in cancer incidence and mortality; importance of, and commitment to, a variety of health concerns; present availability of and future plans for cancer control programs and services; and the development of culturally relevant educational materials.

Data were entered into a EpiInfo6 database and analyzed. To determine statistical and meaningful significance, the chi-square test was applied to normally distributed data and the Mann-Whitney U (non-parametric) test employed when response distributions were skewed.

Table 1.
Sociodemographic Characteristics of Respondents

Characteristics	American Indian Tribes (n=108)	Alaska Native Tribes (n=42)
Ethnicity		
Native American	57%	89%
non-Native	43%	11%
Gender		
Male	26%	23%
Female	74%	77%
Education		
≤ High School	11%	34%
Assoc/BA/BS	45%	43%
MA/MS/MPH	18%	3%
MD/PhD/DDS	9%	0%
Other	17%	20%
Tribal Size		
<200	9%	39%
201-500	17%	44%
501-1000	16%	11%
>1000	56%	6%

RESULTS

Completed questionnaires were received from nineteen percent (n= 42) of AN Tribal communities. This compares with a response rate of 34% (n=108) among AI Tribal Health Directors (x^2 ;$p<.05$). Half (50%) of responding AN communities reported membership less than 275 and 79% reported less than 400 members. (Table 1) A significantly (x^2 ;$p<.05$) greater proportion of AN Health Directors (89%) self-identified as Native American compared to 57% of Directors from the lower 48. Approximately three-quarters (77%) reported being female. Educational levels of the Directors in the two areas also varied. In Alaska 34% reported a high school education only compared to 11% in the lower 48 (x^2 ;$p<.05$). The majority of respondents (39%) in the lower 48 reported having had at least earned a bachelors compared to 23% of their Alaskan counterparts. The number of Tribes accessing care at the respondents clinic was also found to significantly differ between Alaska and the lower 48. In Alaska this ranged from 1 to 50 while the range in the lower 48 was 1 to 75. Approximately half of the AN clinics were accessed by two or fewer different Tribes. This is in contrast to the lower 48 where half reported that their services were utilized by one to four Tribes.

Figure 1: Perceived Trends in Cancer Incidence as Reported by Tribal Health Directors

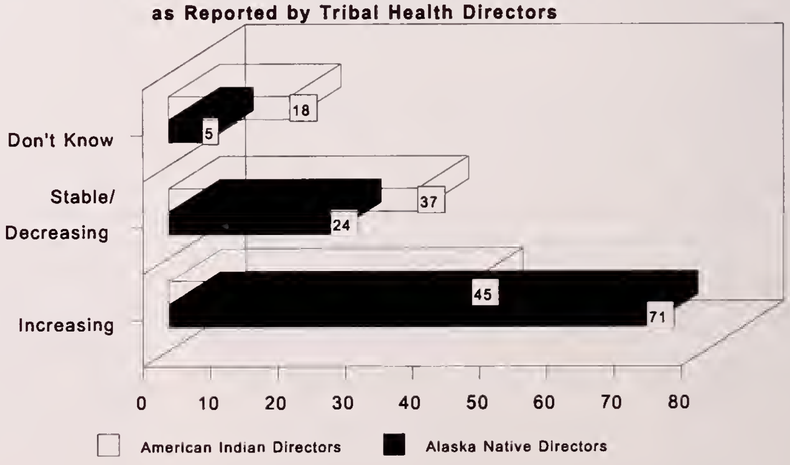
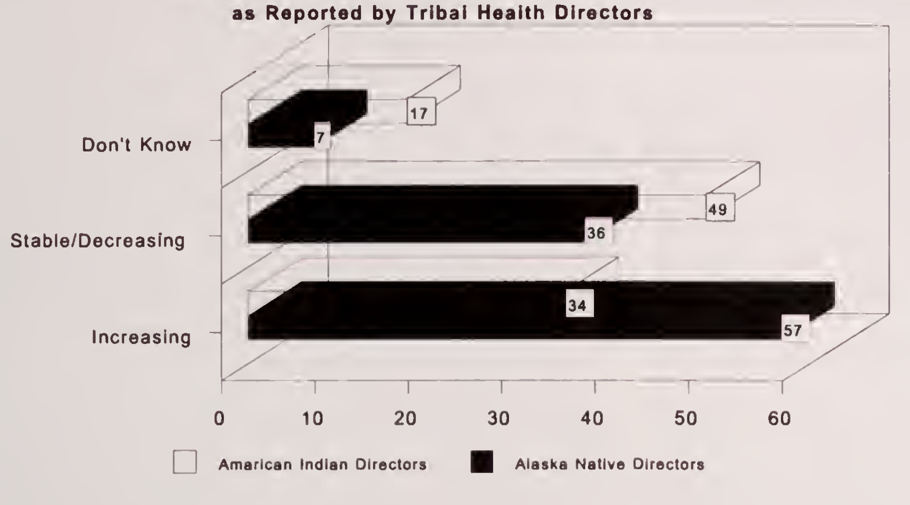


Figure 2: Perceived Trends in Cancer Mortality as Reported by Tribal Health Directors



Tribal Health Directors were asked about their perceptions concerning trends in cancer incidence and cancer mortality rates among their Tribal members (Figures 1&2). Cancer incidence rates were perceived to be increasing (x^2 ;$p<.05$) by a greater proportion of AN Directors (71%) than by AI Directors (45%). Most striking was a greater relative awareness among AN Health Directors with only 5% reporting not knowing what the trend in cancer incidence was compared to 18% of AI Directors. Concerning trends in cancer mortality, 57% of AN Health Directors reported increasing rates compared to 34% of Directors in the lower 48. Significantly fewer AN Directors reported not knowing trends both in terms of cancer incidence (5% vs. 18% of AI) and cancer mortality (7% vs. 17% of AI)

Directors were then asked how important cancer was compared to other health problems experienced by Tribal members. Nearly one-quarter more AN Directors (45.4%) reported cancer as somewhat/much more important than AI Directors (21.2%; x^2, $p<0.05$). No AN Health Director reported cancer to be much less important while 10.6% of AI Directors did. Conversely, 30% of AI Directors report cancer to be somewhat/much less important compared to 9.5% of AN Directors.

Tribal Health Directors were asked to rank their Tribe's commitment to confronting 7 distinct health problems. Health conditions were selected based on their importance as reflected in the literature relative to AI/AN populations. Table 2 presents the relative rankings ascribed to each health condition based on the Health Directors perception of that condition as being their Tribe's highest priority (which is why percents do not total 100%). Cancer was ranked third by AN Directors, based on percent reporting it to be of greatest concern, relative to a ranking of five ascribed by AI Directors (Mann-Whitney U test; p=.0267). The number one priority identified by AN Directors was alcoholism (49%), followed by accidents and injuries (27%), cancer (19.5%), AIDS (12%), diabetes (7%),cardiovascular disease (5%), and tuberculosis (2.5%). In contrast, diabetes (50%) was ranked first by AI Directors followed by alcohol (47%), cardiovascular disease (21%), accidents and injuries (17%), cancer (12%), AIDS (7%) and tuberculosis (7%).

Similar results were obtained when Health Directors were asked to rank a series of health programs in terms of the amount of money or resources that the Tribe allocates to each (Table 3). Cancer was ranked fourth by AN and sixth by AI Directors. In both areas, the greatest proportion of respondents reported allocating the greatest resources to alcohol abuse (ranked first by 62% AI and 47% AN respondents). While 32% of AI Directors ranked diabetes as the recipient of the majority of funds/resources, only 10% of AN Directors responded similarly.

Directors were next presented with a list of cancer control services and asked if their Tribe currently sponsors or directly supports each one. A greater proportion of AI Tribes report providing these services relative to their AN counterparts. Sizable differences are noted for each service. Percent reporting provision of service by tribal community are: mammography (50% AN vs. 82%

Table 2.
Relative Rankings of Various Health Conditions

Health Condition	Rank* Alaska Native Tribes (n=42)	Rank* American Indian Tribes (n=108)
Alcohol	1	2
Injuries	2	4
Cancer	3	5
AIDS	4	6
Diabetes	5	1
Cardiovascular	6	3
Tuberculosis	7	7

* Relative Rankings based on percent of Health Directors reporting each condition as highest priority

Table 3.
Prioritization of Various Health Conditions based on Resources Allocated by Tribe

Health Condition	Rank* Alaska Native Tribes (n=42)	Rank* American Indian Tribes (n=108)
Alcohol & Substance Abuse	1	1
Maternal & Child Health	2	3
Injury Control	3	5
Cancer	4	6
Diabetes Control	5	2
Cardiovascular	6	4
AIDS	7	7

AI); cervical screening (59% AN vs. 80% AI); colorectal screening (33% AN vs. 65% AI); smoking cessation (34% AN vs. 67% AI); and smoking prevention (41% AN vs. 67% AI).

Tribal Health Directors were queried as to what culturally sensitive materials, if any, their Tribe had access to or had developed for their members. Choices covered materials pertaining to breast health, cervical cancer screening, colorectal cancer screening, tobacco control, and general cancer information. Only 24% of AN Health Directors and 35% of AI Health Directors reported having developed any print or video cancer information materials. Those that did, focused primarily on smoking prevention/cessation [9 of 42 (21%) of AN and 31 of 105 (29%) of AI], and breast cancer [8 of 42 (19%) of AN and 32 of 105 (23%) of AI].

DISCUSSION

Alaska Natives at the beginning of this century were reportedly rarely afflicted with cancer. Urquhart (1935) reported on his experiences as a physician practicing for seven years in the Western Arctic (4). He stated that he had never "seen a single case of malignancy in either Eskimo or Indian". This, despite his familiarity with what he called the older, "cancer-age people". Rabinowitch reiterated these sentiments in 1936 (5). He reported seeing but a single suspicious case of cancer, that being a lesion on the lip of a 60 year-old man. Brown (1952) claimed to have reported the first documented case of cancer in an Eskimo (6). Autopsy examination revealed an epithelial mass in the patient's neck probably arising from the thyroid. Rather than attributing the rarity of cancer among Eskimos to immunity, he astutely noted the difficulties in providing medical care to this population during this period and pointed out that the infrequency of cancer may be due to the extremely young age of the Eskimo population, which he cites at the time was predominantly less than 25 years of age.

The earliest organized study of disease in Alaska Natives was presented by Gottmann in 1960 (7). He noted in a review of 103 autopsies during the period 1956-1958, that there were 27 (26.2%) deaths from neoplasms. Arthaud (1970) replicating Gottman's work, reviewed 339 autopsies reported to the Alaska Native Medical Center from 1959 through 1968 (8). He reported that malignancies accounted for 36.9% (n=125) of adult deaths. Schaefer (1975) reported on 180 cases of cancer diagnosed between 1949 and 1974 (9). He noted significantly elevated mortality from cancer relative to the general population. In specific abundance were cancers of the lung, nasopharynx and salivary glands.

Personal observations and case series reports have more recently been supplanted by population-based reports. Lanier et al. (1976) produced the first population-based report on cancer incidence for Alaska Natives covering the years 1969-73 (10). Most recently, Lanier et al. published a monograph on cancer in Alaska Natives covering 1969-88 (11). Data reveal that Alaska Natives present cancer patterns which differ from the U. S. general population and American Indians in the lower 48. Alaska Natives were observed to be at significantly greater risk for cancers of the nasopharynx, salivary gland, liver, gallbladder, kidney and thyroid. Significant deficits were noted for cancers of the prostate, breast, uterus, and melanoma.

Alaska Native elders report that cancer was not a common form of disease among the population in the past. That is no longer the case. Cancer is the leading cause of death among women and second among men. Now, nearly every Native family and community has had first hand awareness of this disease. A review of age-adjusted cancer incidence data for the period 1969 to 1988 reveals that among females, the rate for all sites combined increased from 309.8 per 100,000 in 1969-73 to 414.5 per 100,000 in 1984-88 ($p<0.05$). For males, the age-adjusted rate for all sites increased from 302.9 to 332.7. Between these two periods Alaska Natives also experienced significant increases for cancers of the colon and rectum (females: 74.6 to 89.3; males 60.4 to 61.9); lung (females: 15.2 to 74.8; males: 49.9 to 91.4); and breast (females: 37.9 to 75.6). In terms of mortality, the age-adjusted mortality rate per 100,000, 1989-91, for U. S. All Races was 135.0 relative to 144.5 for Alaska Natives (12).

When Tribal Health Directors were queried if their Tribe maintained statistics on cancer deaths, it was noted that only 14% of AN Health Directors and 31% of AI Health Directors responded in the affirmative. Thus, when posed to evaluate temporal trends in cancer in their community Health Directors related their perceptions based on personal experiences. The impressions of AN

Health Directors were somewhat different than those reported by AI Health Directors. A greater proportion (72%) of AN Health Directors perceived cancer incidence rates to be increasing among their people as opposed to that reported by AI Health Directors (46%). AN Health Directors also demonstrated greater awareness of the cancer problem with only 5% reporting not knowing what the trends were compared to 18% of AI Health Directors reporting not knowing. This variance in Tribal Health Directors perceptions may also be influenced by the high degree of heterogeneity among AI/AN and reflective of true differences in cancer rates by tribal group (12-16).

Tribal Health Directors were next asked to rank (1=highest, . . . 7= lowest) their clinic's commitment in confronting seven different health problems. Several notable differences were evident in comparing the responses from AN Health Directors and AI Health Directors. As noted earlier, AN Health Directors ranked cancer third, while AI Health Directors ranked cancer fifth among the seven diseases. This finding reinforces those significant differences observed among Health Directors for perceptions of temporal trends in cancer rates, as well as the reality of differences in rates. These data indicate that cancer is of greater concern to Alaska Native communities.

IHS data for 1989-91 indicate that while the age-adjusted mortality rate from malignant neoplasms for the overall IHS area (111.2 per 100,000) is 18% lower than the U. S. All Races rate (135.0 per 100,000), the mortality rate for the Alaska area (144.5 per 100,000) is 7% higher than the U. S. All Races rate. There were also differences in the specific cancers identified for action by the health directors. Directors were presented with 7 cancer sites and asked to rank them from greatest to least concern. While there was not much difference in ratings received for cancers of the cervix (35% AN vs. 38% AI), others sites exhibited differences. Many more AI Health Directors were concerned with breast cancer (56% vs. 38% of ANHDs). AN Health Directors, on the other hand, were much more concerned with cancers of the lung (41% vs. 19% of AI Health Directors), colon (20% vs. 15% of AI Health Directors),prostate (15% vs. 8% of AI Health Directors), gallbladder (11% vs. 6% of AI Health Directors) and leukemia (8% vs. 4% of AI Health Directors). These priorities reflect differences in cancer patterns between Alaska and the lower 48.

Approximately equal proportions of AI and AN Health Directors rated alcohol as the number one problem. IHS adjusted All Area age-adjusted alcoholism mortality rate for 1989-1991 was 51.8 (12). This is more than 630% higher than the U. S. All Races rate of 7.1. The rate in the Alaska area is 43.2 or 500% higher than the U. S. All Races rate. Another notable difference is the proportion of Health Directors ranking diabetes as the major health problem. Half (50%) of all AI Health Directors so ranked diabetes as opposed to only 7% of AN Health Directors. Again, according to IHS data, the Alaska area had the lowest age-adjusted diabetes mellitus mortality rate compared to other IHS Areas. The Alaska Area rate of 9.8 was less than the U. S. All Races rate of 11.7 per 100,000 and far less than other IHS Areas which ranged from 26 to 62 per 100,000. Relative to cardiovascular disease, approximately 5% of AN Health Directors ranked it number one compared to 21% of AI Health Directors. This finding also appears to agree with reality. Cardiovascular disease is the number one cause of death in six of eight IHS Areas (3 Areas from the Lower 48 excluded due to underreporting of Indian race on death certificates) (12). Percent of deaths due to cardiovascular disease ranged from 15 to 27 percent. Deaths due to cardiovascular disease was the second most common cause of death in the Alaska accounting for 17% of all deaths. Diseases of the heart account for one-third of all deaths for U. S. All Races. The number one cause of death in the Alaska Area (both sexes) is death due to accidents & adverse effects; accounting for 21% of all deaths. Accidents & injuries was ranked second by AN Health Directors and fourth by AI Health Directors.

As alluded to earlier, cancer was a rare occurrence at the turn of the century among American Indian and Alaska Native communities. While these groups as a whole still experience lower overall cancer incidence rates than other ethnic groups in this country, the differential is diminishing. Over the course of the past several decades, cancer has become a major public health concern. For example, Alaska Natives (both sexes) have the highest age-adjusted (1970 Standard) incidence rates (i.e., number of cases per 100,000) of any racial or ethnic group (e.g., whites, blacks) for cancers of the colon and rectum (62.6), cervix uteri (28), oral cavity and pharynx (16.5), and kidney and renal pelvis (11.2) (16). While increases in cancer incidence should be of paramount concern, attention must also be paid to mortality. When racially-specific data are examined, Alaska Natives (both sexes) have the highest age-adjusted (1970 Standard) mortality rates of any racial or ethnic group for cancers of the colon and rectum (e.g., 24.6 AN, 22.4 for African American and 21.3 for Whites), cervix uteri (e.g., 12.5 AN, 8.7 African American, 3.2 Whites), oral cavity and pharynx (e.g., 8.3 AN, 5.7 African American, 3.3 Whites), kidney and renal pelvis (e.g., 5.5 AN, 2.7 African American, 3.2 Whites), and gallbladder (e.g., 3.8 AN, 0.7 African American, 0.9 Whites) (16). American Indians experience the poorest survival from cancer than any other racial/ethnic group in this country. The five-year relative survival from cancer for all sites combined is 35.2% among American Indians versus 50.3 for Whites. Many of the cancer sites contributing to this dismal picture are amenable to early detection and screening. If we were to approach Healthy People Year 2000 goals, particularly as they relate to screening activities, then we must begin to identify and dismantle barriers and facilitate access and utilization. However, health care providers need to be flexible and plan for the rich heterogeneity of these groups. Not only are there cultural differences between tribal groups, but subtle and sometimes dramatic differences in cancer patterns. Data for American Indian females document that the five leading cancers are breast, cervix uteri, gallbladder, stomach, and colorectum (16). The five leading cancers among Alaska Native females are colorectum, breast, cervix uteri, lung, and oral cavity (11). Alaska Native and American Indian males likewise experience different patterns. The five leading cancers among American Indian males are: prostate, stomach, colorectum, lung, and kidney. Among Alaska Native males the lading cancers are: lung, colorectum, prostate, stomach, and oral cavity. When each of these sites is examined, unusual patterns emerge within the sub-population. For example, Alaska Natives living in the State of Alaska have the highest age-adjusted colon and rectum cancer incidence rate per 100,000 population for both sexes (Alaska 1977-83) in comparison with all other racial and ethnic groups. The Alaska Native rate is 62.6 and the White rate is 52.8. The American Indian (Arizona and New Mexico data) colorectal cancer incidence rate for both sexes is significantly lower (10.2) than the White rate.

When Tribal and IHS Area data are reviewed, even more striking Alaska Native subgroup differences are revealed (17). Among Alaska Native males, colorectal cancer incidence rates are 61.0 (White male rates are 64.5). For Aleut males, the colorectal cancer incidence rate is much higher, 114.8. Other Alaska Native male groups are significantly lower in colon and rectum incidence rates. For example, Eskimo males have a rate of 53.2 and Athapaskan males have a rate of 40.4. In contrast, in the American Indian subgroup, the male colorectal cancer incidence rate for Arizona and New Mexico is 10.06, which is significantly lower (15,16).

Based upon Alaska Area IHS data, Alaska Native women have the highest colorectal female incidence rate of any racial group, 90.2 (African American women have the second highest rate at 45.9) (16). A comparison within the three subgroups of Alaska Native women reveal that Eskimo women have the highest rate at 116.1. Athapaskan women have the second highest rate, 96.2. Aleut females have an incidence rate of 89.9. In comparison, the White female rate is 44.9 and the American Indian female rate from Arizona and New Mexico is exceptionally low at 10.0 (15).

Furthermore, gender comparisons in this group illustrate unusual variability. For example, the colorectal cancer incidence rate for Eskimo females (116.1) is over twice the rate for males (53.2). This is also true for Athapaskan females (96.2) compared to males (40.4). On the other hand, Aleut females (89.9) have a lower rate than Aleut males (114.6) (15).

Differences between Alaska Native and American Indian cancer patterns may not be that surprising given the large geographic areas encompassed by each group and the diversity of Tribal groups they encompass. Numerous examples of tribal heterogeneity in terms of cancer risk abound in the literature (12-19). Findings from this survey underscore the need to develop and expand cancer control programs responsive to the regional needs of Tribal communities. Moreover, greater effort should be made to increase Tribal awareness to the cancer threat. As Tribal communities make gains in life-expectancy, so too will they make gains in cancer incidence.

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Smoking Cessation Products and Programs

Harry A. Lando, Ph.D.(1)

Smoking cessation programs have improved significantly over the past 30 years. Hundreds of studies have examined formal approaches to smoking cessation. More detailed reviews of these studies are available elsewhere (1,2). Several types of options are available. There are face-to-face programs which have been offered primarily in small groups and which are most commonly based upon behavioral principles. There are smoking cessation aids and products of which nicotine replacement is currently the only category recognized by the Food and Drug Administration as effective. Other popular approaches have included hypnosis and acupuncture. Programs offered by the voluntary health organizations and by commercial providers have tended to emphasize behavioral techniques.

BEHAVIORAL TREATMENT TECHNIQUES

Smoking and other tobacco use was seen for many years as essentially a learned activity. More recently smoking has become recognized as a physical addiction (3). Behavioral aspects of smoking are still seen as critical, however. One behavioral treatment approach involves aversion. Positive results have been obtained with rapid smoking (e.g., requiring smokers to take a puff every 6 seconds for as long as they can tolerate the procedure) (4,5). Concerns have been expressed about the potential risks of rapid smoking and about its high level of unpleasantness, however. Reduced aversion techniques have shown promise including "focused smoking" in which the patient smokes at a regulated but slower rate and smoke holding in which patients hold cigarettes in the mouth and throat while breathing through the nose (6,7).

Additional behavioral techniques have included contingency contracting in which participants typically self-administer rewards or punishments depending upon abstinence. Most current behavioral approaches emphasize coping techniques as part of multicomponent intervention (8,9). Participants develop a repertoire of behavioral and cognitive activities to counter smoking in high-risk situations.

Nicotine fading has been used as a nonaversive preparation technique for cessation. Gradual reduction in numbers of cigarettes does not appear to work very well beyond a common "stuck point," often 10 to 12 cigarettes per day (10). An alternative is to switch brands over several weeks or to use commercially available nicotine reduction filters to progressively lower rated tar and nicotine levels (11,12). Results for nicotine fading have been mixed, with some studies showing no effect.

The most successful behavioral programs have incorporated multiple interventions. Emphasis has been placed both upon initial preparation for quitting and longer-term maintenance. Long-term abstinence rates for these multicomponent treatments have approached 50 percent (8, 7), although more recent outcomes have tended to be less favorable (13).

A focus of many comprehensive behavioral programs is upon relapse prevention following the quit date (14). Slips are viewed as a natural part of the learning process in quitting. Results for relapse prevention have been mixed, but the overall findings generally do not reveal significant treatment effects. Lando et al. (15) achieved positive results in relapsed smokers using two brief supportive telephone calls. Self-reported 6-month abstinence rates were 13% for those who received telephone support and only 6% for those who did not receive support. Differences between telephone support and control conditions were no longer significant at 18 months, however. In a subsequent study, more intensive telephone support also was found to significantly increase recycling among relapsers. Two years following the initial intervention, 26% of those who had relapsed in the telephone support condition were abstinent, as compared to 18% of relapsers who did not receive telephone support.

HYPNOSIS AND ACUPUNCTURE

There are numerous approaches to smoking cessation that are not primarily behavioral. Two commonly advertised methods include hypnosis and acupuncture.

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(1) Division of Epidemiology, University of Minnesota, 1300 South Second St., Suite 300, Minneapolis, MN 55454-1015. Preparation of this article was facilitated by Grant #R01HL48121 from the National Heart, Lung, Blood Institute. Much of the content of this article was condensed from Chapter 11 of *Nicotine Addiction: Principles and Management*, edited by C. Tracy Orleans and John Slade, New York: Oxford University Press, 1993. This chapter was written by Harry A. Lando and was entitled "Formal Quit Smoking Treatments."

Unfortunately, there are few good studies of either of these methods and overall results tend to be disappointing. These methods appear to pose little risk, however, and some patients may benefit from expectations of successful outcome.

PHARMACOLOGIC APPROACHES

By far the most intensively studied pharmacologic methods have involved nicotine replacement. The largest number of studies have been done on nicotine gum, followed by nicotine patch. Recent work has been done with other forms of nicotine replacement including nicotine nasal spray and nicotine inhalers. Although these newer products appear promising, few published studies are available. Both nicotine gum and nicotine patch improve treatment outcomes. The patch may be preferable for most patients partly because the results of patch studies have tended to be somewhat more positive than those for gum and partly because compliance tends to be better for patch (16). Meta-analyses have indicated that nicotine gum increases abstinence by about 60% on the average compared to placebo controlled trials. Meta-analyses of nicotine patch studies show an approximate doubling of success rates. However, to date no study has directly compared the effectiveness of nicotine gum and patch. Currently nicotine replacement products are available only by prescription, but the Food and Drug Administration has approved 2 mg gum for over-the-counter use.

Some evidence suggests that clonidine may be effective in facilitating smoking cessation, especially among women, but the findings have not been consistent. Several studies have shown no effect for clonidine in either women or men. Clonidine is not approved by the FDA for smoking cessation. It does appear to diminish cravings, but its value is limited by unpleasant and sometimes severe side effects including dry mouth, fatigue, and dizziness. Some anxiolytics have been demonstrated to reduce withdrawal and may be useful especially for depressed smokers. However, data are very limited and the FDA does not recognize these drugs for smoking cessation.

VOLUNTARY HEALTH ORGANIZATIONS AND COMMERCIAL PROGRAMS

Voluntary Health Organizations

Major nonprofit organizations offering smoking cessation programs include the American Cancer Society, the American Lung Association, and the Seventh-Day Adventist Church. The American Cancer Society FreshStart program consists of four, one-hour group sessions. Content of this program has been recently revised. The American Lung Association program consists of seven 90-minute to 2-hour group sessions in addition to an initial orientation over a seven-week period. Both programs include considerable behavioral content.

Lando et al. (17) compared these two methods. Abstinence rates at 12-month follow-up were 19% and 12% for the American Lung Association and American Cancer Society programs, respectively. It should be noted, however, that the American Cancer Society groups are considerably less intensive and therefore presumably less costly to run. The absence of a specific target date for quitting in the American Cancer Society method may detract from effectiveness.

The Seventh-Day Adventist Church has sponsored Five-Day-Plans which are the oldest of the nonprofit programs (18). These programs do not emphasize cognitive-behavioral strategies but instead focus on psychological and physical components of nicotine dependence. An estimated 14 million smokers in more than 150 countries have attended Five-Day-Plans. The program is held on consecutive days and includes five 90-minute to 2-hour lectures. Recently the Five-Day-Plan has been revised to 8 sessions over a 3-week period. This program (now called the "Breathe-Free Plan to Stop Smoking") also offers supportive telephone calls 1 week, 3, 6 and 12 months after completion of the program.

Schwartz (2) reported summary outcome data based on 14 evaluations of the Five-Day-Plan. These data indicated a median 1-year abstinence rate of 26%. Results might be less favorable for the current population which appears to consist of a greater proportion of harder core smokers. Outcome data for the revised program currently are not available.

Commercial Programs and Products

A number of commercial programs are available, usually concentrated in larger metropolitan areas. Most programs tend not to be highly profitable and therefore do not remain active. Smokenders, Schick Smoking centers, Smokeless, and Smoke Stoppers are among the best known national programs. In evaluating commercial methods, it again appears that the most successful are those that include multicomponent cognitive-behavioral techniques. Some commercial treatments claim extremely high rates of success. Few external evaluations of proprietary methods are available, however. There is no clear evidence that commercial clinics are more effective than those offered by other organizations. Because commercial programs cost more they may attract more committed smokers. Furthermore, a sizable clinic fee may represent an additional motivation for quitting. New products are continually being introduced as aids to smoking cessation. Currently, none of these products are recognized as effective.

SELF - HELP

Self-help interventions for smoking cessation can consist of anything from purely self-help to rather intense, but still self-administered methods. The exact definition of self-help can be somewhat ambiguous. Self-help interventions most often consist of written materials. A major emphasis of these materials is usually on coping strategies, especially in high risk situations. Self-help materials also are available in other formats including audio and video tapes. Some promising results have been obtained with computerized programs that attempt to tailor cessation messages to the individual smoker. Overall, self-help methods in isolation have had limited impact in improving abstinence outcome. Outcomes have been improved, however, when self-help approaches have been supplemented with additional intervention, most notably nicotine replacement and supportive telephone calls.

EVALUATION STANDARDS

It is increasingly important that health professionals be well informed about smoking cessation services and products. They should be skeptical in evaluating claims of effectiveness. Program and product evaluations should be based on a minimum of 6-month and preferably 12-month follow-up. Providers may not deliberately misreport their abstinence data (although this does sometimes occur), but they may present very misleading figures. Thus some programs report end-of-treatment success, based on outcomes only a few days after a target quit date. End-of-treatment abstinence figures of 60% abstinence can translate into 1-year outcomes as low as 5-10%.

Participant attrition also must be considered (19). Programs can dramatically manipulate their outcomes by failing to count nonrespondents to follow-up. Assume, for example, that a program attracts 100 individuals of whom 50 complete the treatment, 30 respond to the follow-up, and 20 report abstinence. Claimed abstinence based on these results could be as high as 67%, whereas 20% abstinence would be a more valid outcome figure.

Resources for additional information are available both locally and nationally. Local resources are listed elsewhere in this issue and include the American Cancer Society, the American Lung Association, and the state health department. The National Cancer Institute provides a toll-free information line (1-800-4CANCER). Information and materials also are available through the U.S. Office On Smoking and Health (1-770-488-5705), the American Medical Association (1-312-464-5000), and the American Society of Addiction Medicine (1-301-656-3920).

SUMMARY AND CONCLUSIONS

Behavioral treatment techniques have facilitated smoking cessation, with intensive multicomponent interventions sometimes producing long-term abstinence rates approaching 50%. There is little evidence that either hypnosis or acupuncture are effective. Both nicotine gum and nicotine patch significantly improve treatment outcomes, although patch is easier for patients to use correctly. Self-help programs may be of benefit, however, smoking cessation products other than nicotine replacement show little evidence of effectiveness. Health professionals should be informed consumers and should be skeptical in evaluating claims for commercial programs or products. Additional information and materials are available from a number of sources including the National Cancer Institute, the U.S. Office on Smoking and Health, and the voluntary health organizations.

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(continued from page 58 - Paralytic Shellfish Poisoning)

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Prevention of Ovarian Cancer with Bilateral Oophorectomy at Time of Hysterectomy

Patricia A. McGuire, R.N., O.C.N.(1)
Beth H. Conklin, R.N., B.S.N., O.C.N.(1)
Dale I. Webb, M.D.(1)

ABSTRACT

A review of all patients with a diagnosis of ovarian cancer for five years (1989-1993) in the military hospital, the public health hospital and the two private hospitals in Anchorage, Alaska was conducted. Fifty-eight women were found with a diagnosis of advanced ovarian cancer. These charts were reviewed to determine the incidence of prior hysterectomy with preservation of ovaries. Of the fifty-eight, thirteen (22%) had prior hysterectomies with the ovaries left in place. Review of the literature supported this statistic in similar studies. Ovarian cancer has such a poor prognosis that prevention is in order. The data demonstrate that ovarian cancer could have been prevented in a significant number of these patients had they had bilateral oophorectomies at the time of hysterectomy. With the relative safety of hormonal replacement therapy, a significant way to prevent ovarian cancer is available—bilateral oophorectomy.

INTRODUCTION

The incidence of ovarian cancer has increased over the past decade (1). Women currently face a 1 in 70 chance of developing ovarian cancer (2), the 6th most common cancer in the United States and the 4th most common cause of female deaths, ranking behind lung, breast and colon (2). The cause of ovarian cancer is unknown. Although numerous reports have linked ovarian cancer with environmental factors (such as diet, viruses, and industrial agents) and reproductive history, these associations are not clearly causal. Increased risk profiles have been demonstrated with particular genetic and familial syndromes. Defining and differentiating between "Familial" and "Hereditary" ovarian cancer is difficult, and further data is needed to make progress in the area of early diagnosis. While CA-125 is elevated in 80% of ovarian tumors, it may not become elevated until the ovarian cancer is Stage-III. Likewise, pelvic ultrasonography is unproven in early detection (3). New treatments of ovarian cancer (Taxol and high-dose chemotherapy with marrow or stem cell support) have not yet increased patient survival. This is probably due to the advanced stage of the disease at the time of diagnosis. Therefore, prophylactic oophorectomy must be considered.

METHODS

A review of all patients with a diagnosis of ovarian cancer for five years (1989-1993) was done at all of the hospitals in Anchorage (2 private, 1 military, and 1 public health). Fifty-eight patients were identified with a histologically proven diagnosis of advanced ovarian cancer. Of these, thirteen patients (22%) had prior hysterectomies with at least one ovary left in place. The reasons for prior hysterectomies were benign disease or hypermenorrhea. The significant information on these thirteen patients was:

— age at diagnosis of ovarian cancer:
    28 - 74 years -- median 58.3 years
— age at time of hysterectomy:
    26 - 45 years -- median 36.5 years
— length of time between hysterectomy and diagnosis of ovarian cancer:
    4 - 36 years -- median 21.5

Of these patients, none had a family history of ovarian cancer. Two patients had history of cancer in their family, but of unknown types. Two patients had prior breast cancer and two had no children. No talc exposure was identified. None of these patients appeared to have significant risk factors.

All thirteen patients had Stage-III or IV disease at time of diagnosis. All of these patients received chemotherapy. Currently, seven patients have expired (survival 1 - 4 years), three are lost to follow up, three have recurrent disease and are currently being treated with only partial responses.

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(1) Providence Medical Office Building, 3340 Providence Drive, Suite 352, Anchorage, Alaska 99508-4627.

DISCUSSION

Hysterectomy is the most common major operation in the United States (8). The incidence of hysterectomy approaches 60% by the time a women reaches 65 years old (7). The morbidity and mortality is very low for both vaginal and abdominal approaches; with vaginal hysterectomies being obviously less invasive (9). The incidence of oophorectomy is 20-30% of all women undergoing hysterectomy (7). It is clear the post hysterectomy ovarian function is not fully understood. Possibly half the patients after simple hysterectomy develop decreased ovarian production (7).

A review of the literature demonstrates that the majority of gynecologists favor preservation of ovaries in premenopausal women (4,5). There is more controversy for peri and post menopausal women with possible regional influences in the data. However, most favor ovarian preservation, and in fact, state that no scientific arguments justify routine oophorectomy at time of hysterectomy (5). At best, we find those who recommend oophorectomy at time of hysterectomy suggest such on a case by case basis (6).

Numerous reasons are given for ovary preservation. The obvious being to keep intact hormonal production, with the concern of osteoporosis, heart disease and general well being of women. The post menopausal ovary also secretes androgens which may be beneficial.

Some authors have theorized that tubal ligation and hysterectomy may reduce the risk for ovarian cancer, possibly due to preventing vaginally induced talc (10). However, talc association was unclear and conclusions from their data concerning the incidence of ovarian cancer due to talc exposure were unfounded.

In the studies reviewed, the percent of patients developing ovarian cancer after simple hysterectomy varied widely from 3.6% to 18%(4,11). (See table) The lower percentages were from older studies. When the data was collected from hysterectomized patient populations, the years of follow up were not indicated. Short follow up may not include all those women who eventually developed ovarian cancer. Since the average age of all ovarian cancer patients at time of diagnosis is known to be 65 years, (2) data would not be mature for a number of years in some studies (not until at least the year 2000 for the Hankinson Study) (10). The Scottolini Study in Hawaii showed 18% of ovarian cancer patients had prior hysterectomies (11). The McGowan Study done in Washington D.C. showed a 14% rate in the late 1980s (8), compared to a similar study done in the same area in the mid-70s when only a 1% rate of women with ovarian cancer were found to have had prior hysterectomy. Is this due to the increasing incidence of ovarian cancer or rather, as the author suggests, to a shift in practice patterns as a result of concern for osteoporosis and women's health issues (where past practice patterns were to remove normal appearing ovaries in women 40 and over during hysterectomy; and now — in the 90s — more women are keeping their ovaries (8))?

Certainly, because of the severe risks of osteoporosis and vascular disease, all patients considered for prophylactic oophorectomy must be advised of the need for estrogen replacement therapy. Premenopausal women who refuse estrogen should not be considered for prophylactic oophorectomy (12).

Finally, in a survey of fellows and members of the Royal College of Obstetricians and Gynaecologists (13), the authors concluded that the potential role of prophylactic oophorectomy in ovarian cancer prevention is underestimated by surgeons due to a lack of knowledge about this subject. However, this seems not the case in Alaska (see accompanying article on survey results of Alaska's gynecologists).

SUMMARY

Ovarian cancer continues to be difficult to diagnose and most patients have Stage-III or Stage-IV disease when diagnosed. Only 25-30% survive five years (11). With such disheartening statistics, methods to prevent this disease must be considered. This study and others demonstrate the need for gynecologists to reconsider oophorectomy at the time of hysterectomy — in all patients. With the relative safety of replacement hormonal therapy, there is little reason to preserve ovaries.

Table 1.

Review of Literature

	Authors	Year of Study	Incidence
1.	Terr, J. Barber, H.	1967	5.1%
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6.	Thorp, D.	1951	3.6%
7.	Pemberton, F.	1960	6.7%
8.	Gibbs, E.	1971	11.9%
9.	Scottolini, A.	1986	18.0%
10.	Sightler et al	1990	12.6%
11.	McGowan, L.	1987	14.1%
12.	McGuire et al	Present Study	22.0%

Table of Studies Showing incidence of ovarian cancer after hysterectomy.

(continued on page 81)

A Survey of the Practice of Prophylactic Oophorectomy by Gynecologists in the State of Alaska

Beth H. Conklin, R.N., B.S.N., O.C.N.(1)
Patricia A. McGuire, R.N., O.C.N.(1)
Pamela L. Weiler, R.N.(1)
Dale I. Webb, M.D.(1)

SUMMARY

A questionnarie was sent to Alaskan gynecologists to evaluate their practice in regard to prophylactic oophorectomy at the time of hysterectomy. At issue is the possible benefit of prophylactic oophorectomy in the prevention of ovarian cancer. Sixty-seven questionnaires were sent and 42 (63%) were returned completed. The percentage who said they would remove apparently normal ovaries at the time of hysterectomy in premenopausal women is as follows: at 35-39 years, 5% ; 40-44, 35%; 45-49, 81% and over 49 years, 95%. In postmenopausal women, 97% would recommend removal of the ovaries. 29% of respondents believe that more than 10% of ovarian cancers could be prevented by prophylactic oophorectomy at the time of hysterectomy.

INTRODUCTION

Ovarian cancer is the leading cause of death from gynecologic malignancies in the U.S. In 1995, it is estimated that 26,600 new cases will be diagnosed in the U.S. and 14,400 deaths per year will occur (1). Screening and detection of this disease remains difficult and the majority of women (70%) have advanced disease at the time of diagnosis. The 5 year survival rate in this group is 15-20% in spite of advances in surgery and chemotherapy. Thus, the prognosis has remained largely unchanged for 30 years. Although this disease can be cured when detected early, historically the only tools for detection have been the history and physical examination. A randomized trial has been undertaken by the National Institutes of Health which will help answer the question as to the feasibility and value of screening detection using existing technology, i.e. transvaginal ultrasonography and serum biomarkers (CA-125). At present though, oophorectomy remains the only proven method for prevention of ovarian cancer.

In reviewing patients with ovarian cancer, we questioned whether there was a significant percentage who would have benefited from oophorectomy at the time of hysterectomy. The practice of removing apparently normal ovaries is controversial and there are no accepted criteria in premenopausal women. Given our interest in prevention of ovarian cancer, we set out to investigate practices of prophylactic oophorectomy in the Alaskan gynecology community.

METHODS

A questionnaire designed by Jacobs and Oram (2) for a survey done in the U.K. was modified with permission of the authors. After obtaining the names of practicing gynecologists from the Alaska State Medical Association, we mailed each the questionnaire shown in table 1. These physicians were from all geographical locations around the state. The completed questionnaires were returned by mail , fax or hand delivery. All responses are kept confidential.

RESULTS/DISCUSSION

Of the 67 questionnaires sent, 42 (63%) were returned. The results are shown in Tables 1 and 2.

Question 1 addressed the influence of age and menopausal status on the practice of prophylactic oophprectomy. Loss of ovarian function prematurely must be weighed against the lifetime benefit of reduced risk of ovarian cancer. Questions regarding compliance in hormone replacement therapy and the unknown role the ovaries play in the hormonal milieu are problematic for recommending prophylactic oophorectomy. There is majority agreement as to whether or not a woman undergoing hysterectomy should retain ovaries in every age group and menopausal state with the clear exception of the premenopausal woman, ages 40-44. In this age group, 36% of the physicians polled would advocate removal of the ovaries, 45% would not, and 19% are

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(1) Providence Medical Office Building, 3340 Providence Drive, Suite 352, Anchorage, Alaska 99508-4627.

Table 1.

1. When performing an abdominal hysterectomy for benign disease, is it your usual practice to remove apparently normal ovaries in;

	Yes	No	No Answer/Undecided
Premenopausal women aged 35-39 years	(5%)	(83%)	(12%)
Premenopausal women aged 40-44 years	(36%)	(45%)	(19%)
Premonopausal women aged 45-49 years	(81%)	(5%)	(14%)
Premenopausal women over 49 years	(95%)	(2.5%)	(2.5%)
Postmenopausal women	(98%)	(0)	(2%)

2. When deciding whether or not to remove apparently normal ovaries is your decision influenced by the following?

	Yes	No	No Answer
Family history of gynecologic cancer	(90.5%)	(9.5%)	
Past history of breast cancer	(71%)	(26%)	(3%)
Parity	(12%)	(86%)	(2%)
Contraceptive practice	(10%)	(88%)	(2%)

3. When you perform a prophylactic oophorectomy in a premenopausal woman is it your practice to prescribe hormone replacement therapy?
YES, ORAL (100%) YES, IMPLANT (0) NO (0)

4. Do you consider unilateral oophorectomy to have a role in the prevention of ovarian cancer?
YES (5%) NO (95%)

5. Do you routinely discuss the question of prophylactic oophorectomy with your patients pre-operatively?
YES (95%) NO ANSWER (5%)

6. What percentage of ovarian cancers in the U.S. do you think could be prevented by prophylactic oophorectomy at the time of operation for benign disease?

% prevented	Respondents
1 - 10%	41.8%
11 -20%	21.4%
21- 30%	2.4%
Over 30%	4.8%
No Answer	28.6 %

7. Would you perform prophylactic oophorectomy as a primary surgical procedure in women with a strong family history of ovarian cancer?
YES (85.7) NO (9.5%) NO ANS (4.8%)

8. How long have you been in practice?

1-5 years	26%
6-10 years	31%
11-20 years	31%
> 20 years	12%

9. Approximately how many vaginal hysterectomies do you perform in one year? t=514 avg. 13/yr
What percentage of these include bilateral salpingoophorectomies? Table 2

10. Approximately how many abdominal hysterectomies do you perform in one year? t=696 avg. 17/yr
What percentage of these include bilateral salpingoophorectomies? Table 2

Table 2.

Question 9	Estimated Vaginal Hysterectomies Performed Annually (514)	Number of Gynecologists (40)
	1-10	26
	11-20	8
	21-30	4
	>30	2
	Percent of Vaginal Hysterectomies with BSO	
	1-25%	18
	26-50%	15
	51-75%	5
	76-90%	2
Question 10	Estimated Abdominal Hysterectomies Performed Annually (696)	Number of Gynecologists (40)
	1-10	15
	11-20	18
	21-30	2
	31-40	3
	>40	2
	Percent of Abdominal Hysterectomies with BSO	
	1-25%	2
	26-50%	16
	51-75%	11
	76-90%	10
	>90%	1

undecided. Interestingly, this is compared with results from the U.K. survey in which only 2% would recommend prophylactic oophorectomy in this age group. There is consensus in all other age groups. In Alaska, 83% of the physicians polled would favor retaining ovaries in the premenopausal woman under the age of 40, while 81% and 95% would opt for removal of the ovaries in the 45-49 and over 49 year olds, respectively. In the post menopausal woman essentially all respondents would favor removal of the ovaries.

A majority of physicians would be influenced by a family history of gynecologic cancer (90.5%) and a past history of breast cancer (71%), while parity and contraceptive practice influenced a minority (12% and 10%). Women at higher risk for developing the most common histologic type of ovarian cancer are those with a family history of ovarian cancer (5% lifetime risk compared with 1.4% in the general population) and women with an initial primary malignancy of the breast, colon or endometrium (3,4). Clearly established protective factors include more than one full-term pregnancy (with declining risk for each pregnancy), oral contraceptive use and breast-feeding, all of which tend to reduce the number of ovulations. Tubal ligation may also be a protective factor (5).

All respondents would prescribe hormone replacement therapy after oophorectomy. The majority (95%) did not consider unilateral oophorectomy to play a role in the prevention of ovarian cancer. The majority (95%) would discuss the issue of prophylactic oophorectomy pre-operatively with their patients.

The range of answers to question 6, which asked for an estimate of the percentage of ovarian cancer preventable by prophylactic oophorectomy at the time of operation for benign disease, varied greatly. 42% estimated this percentage at 10% or less, and 28% were unable to answer. 28.6% thought ovarian cancer prevention would be greater than 10%. Review of ovarian cancer patients with a previous hysterectomy suggests prevention potential at a rate of 11.9% to 18% (6,7). Our study (McGuire) found this rate to be 22% in a five year period in Anchorage. Thus it would appear that the correct answer is probably at least 10% and maybe as high as 22%. Of interest is that the average age at hysterectomy in the Anchorage study was only 37.7 years old. In this survey, only 5% of Alaskan gynecologists would recommend prophylactic oophorectomy in patients so young.

Question 7 asked whether or not the practitioner would perform a prophylactic oophorectomy as a primary surgical procedure on a woman with a strong family history of ovarian cancer and 85% of the respondents indicated that they would. Among low risk women, characterized by reporting no family history of ovarian cancer, having had three or more term pregnancies, or taking oral contraceptives for 4 or more years, the incidence of ovarian cancer is 5 cases per 100,000

Table 3.
Approximation of Abdominal/Vaginal Hysterectomies (with percent of oophorectomies) Performed in One Year

Years in Practice	Number of Abd Hyst	With BSO	Number of Vag Hyst	With BSO
2-5	10-15	40-50%	5	20%
	10	100%	10	50%
	10-15	50%	2-3	0
	4-5	75%	12-15	50-75%
	20	70%	5	60%
	10-20	50%	20	10%
	5	60%	2	50%
	15	80%	10	25%
	15	50%	20	90%
	2-3	50%	30	50%
	5	90%	5	50%
6-10	20	70%	10	50%
	15	30%	10	15%
	10	50%	30	20%
	12	50%	4	25%
	30	50%	10	50%
	5	90%	24	10%
	15	75%	8	75%
	12	30%	4	25%
	15-20	80%	5-10	5%
	10	80%	0	0%
	20	40%	5	50%
	20	40%	5	0%
11-15	5-7	80%	3-5	50%
	10	95%	15	30%
15-20	8-10	50-75%	8-10	50%
	40	90%	30	80%
	2	90%	2	75%
	40	50%	25	0%
	25	30%	10	10%
	12	50%	2	0%
	70	60%	10	20%
	20	90%	12	0%
	10	20%	6	33%
20+	50	75%	20	50%
	7-10	70%	3-4	50%
	40	75%	20	40%
	8	75%	50	75%
	20	80%	20	50%
	20	50%	50-60	50%

woman-years, compared to 78 cases per 100,000 woman-years among women reporting that a sister or mother had ovarian cancer (8). This is striking evidence for the need to remove ovaries in these women and Alaskan gynecology practice reflects such.

Questions 9 and 10 asked for an approximate number of hysterectomies performed on an annual basis in the respondents' practice and then for a percentage of how many of those included oophorectomy (Table 3). A total of 514 vaginal hysterectomies were approximated in one year, with the majority of the physicians (78.5%) including oophorectomy less than half the time. Abdominal procedures, estimated number 696, included oophorectomy more frequently, with most including oophorectomy in 25-90% of their procedures. Only 2 respondents performed oophorectomy during abdominal hysterectomies less than 25% of the time. These numbers, while suggesting that oophorectomy is performed at a higher rate during abdominal hysterectomies, suggest that Alaskan gynecologists are skillful at oophorectomy with vaginal hysterectomy as well.

ACKNOWLEDGMENTS

LuAnn Webb for her assistance in statistical analysis.

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Bartlett Memorial Hospital Celebrates 25 Years

Kenneth W. Moss, M.D.

May 1994. Front entrance of Bartlett Memorial Hospital

May 1996. Bartlett Memorial Hospital

Bartlett Memorial Hospital. Photos courtesy of Kenneth W. Moss, M.D.

Emergency Entrance of Bartlett Memorial Hospital

Bartlett Memorial Hospital will celebrate 25 years of service to the community of Juneau and Southeast Alaska this summer.

Named for U.S. Senator Bob Bartlett, one of Alaska's first two senators, the hospital opened its doors on August 19, 1971. The hospital is owned by the City & Borough of Juneau and operates with an administrative services contract through Quorum, Inc. Hospital Administrator Robert Valiant and Chief of Medical Staff, Allan G. Schlicht, MD, work with a nine member hospital board, coordinating and managing the many services available to the community.

There are 52 physicians on the active medical staff and a consulting and courtesy staff. In addition to primary care specialties of family practice, pediatrics, and internal medicine, there are three general surgeons and three orthopedic surgeons on active staff. Other specialities include emergency medicine, ophthalmology, otolaryngology, plastic surgery, urology, radiology, gynecology, dermatology, physiatry, and neurology. Additions to the radiology department have been made over the years and now include CT-scan, MRI, ultrasonography, and nuclear medicine.

Bartlett Memorial Hospital is a 51 bed, JCAHO certified general medical and surgical hospital serving a regional population of about 50,000, and provides additional direct and supportive services for large numbers of cruise ship passengers and other tourists each year.

Approximately 13,000 residents and visitors were seen as patients in the Emergency Department in 1995. There are around 500 babies born each year at BMH and over 2,800 births since 1990.

Sexually Speaking . . .

Mary Cavalier, M.S.[1]

FROM LOVER TO CAREGIVER . . .

A Series of the Effect of Illness on Intimacy

Part One

Recently, I have been consulting on the Oncology Unit at Alaska Regional Hospital. As I listen to the cases being presented, I am reminded of an often overlooked aspect of treatment — the sex life of the couple.

In this series, I will be discussing the effects of pharmaceutical treatments, the physiological, and psychological effects of treatment. In this part I will be discussing the effect of the transition from lover to caregiver.

Dependency

Serious illness can have a profound impact on a relationship. The increased dependency on a partner can trigger abandonment issues which go back to childhood. In turn, the partner may experience feelings of inadequacy of "not being enough." These fears often are expressed in indirect ways such as irritability, belittling, anger, feelings of hopelessness and depression.

Sometimes an intervention can be simply the doctor bringing up these potential reactions when discussing the physical care which will be needed at home. By naming the fears, it can minimize the impact so it won't be a total surprise.

Romance and Bed Pans

Let's face it, there is nothing romantic about vomit and bed pans. The impact of taking care of a loved one with a serious illness often comes in layers of awareness of how much life is changing. The caregiver may grieve the loss of the sexual intimacy, and may feel guilty for having sexual feelings. After all, aren't we supposed to be focusing all our energy on the ill one? One may feel selfish for having sexual feelings.

Frank discussion between care provider and the couple about the changes of the sex life may enable the couple to discuss the range of feelings of the caregiver. It's important to acknowledge the fact that the caregiver may experience sexual desire. Giving permission (within the range of moral beliefs of the couple) for the caregiver to masturbate can be very freeing for the couple.

. . . there is nothing romantic about vomit and bedpans.

Caregiver Burnout

Everybody has a limit to being there for someone else. A person can feel a paralyzing amount of guilt in needing time off from the patient.

It is important to educate the caregiver on self care techniques at the onset of the illness. Prevention is the best medicine. Give the caregiver permission to take time to him/herself, or to get help to come in once in awhile.

I have dealt with situations where the ill person will not allow another to provide care. It is imperative that the doctor firmly state that that is not an option. Sometimes the patient needs to be reminded that caregivers can also get sick unless they take care of themselves. Caregivers cannot truly take care of others until they can nurture themselves.

Conclusion

In conclusion, serious illness changes the intimacy between two people. A lot of problems can be addressed at the beginning by communicating the range of reactions which may occur. Some examples of these are the fear of abandonment and feelings of inadequacy, feelings of guilt for having sexual feelings, and giving permission for self-pleasuring, and finally, caregiver burnout and the need to nurture the self from the very beginning.

Part 2 of this series will be the pharmacological effects of sexuality in the treatment of serious illness. Till then have a great summer!

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(1) Mary B. Cavalier, M.Ed., Counselor, Educator, Private Practice.

From Out of the Past. . .

Gloria Park, M.D.

The "Parron Report" is often mentined in articles and data about Alaska's health so for interest and comparison the following graphs are from that report which was a survey done at the request of the Department of Interior. The chairman of the team was Thomas Parran:

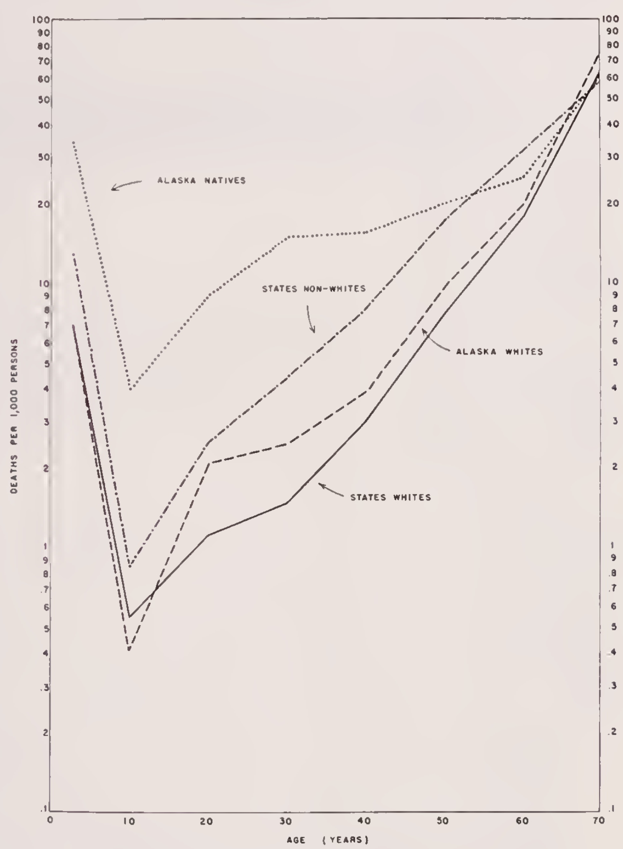
"Alaska's Health"

by the Graduate School of Public Health,
University of Pittsburgh - 1954

Alaska Department of Health, Juneau, Alaska
Section of Preventive Medical Services

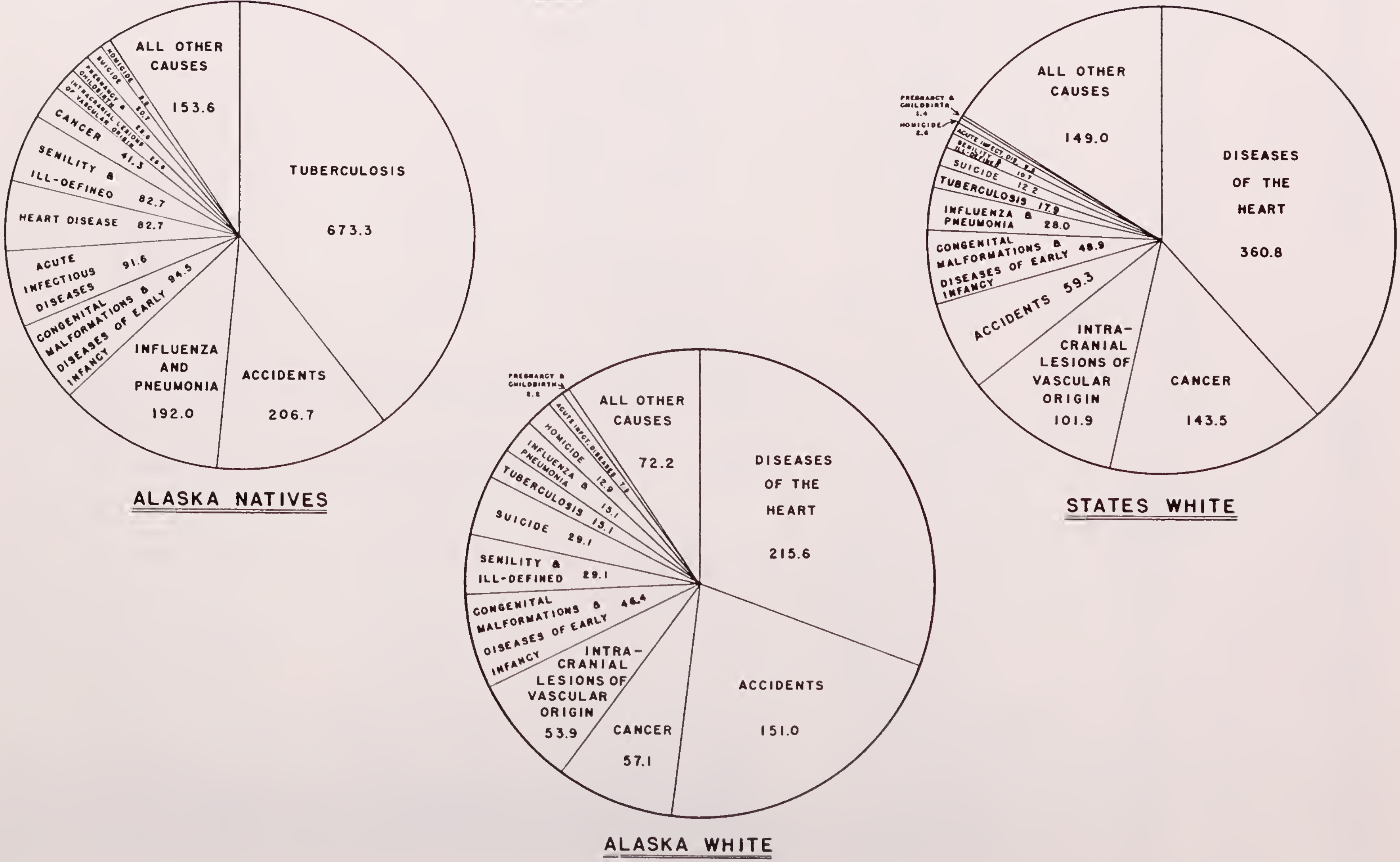
Epidemiological Report
Fiscal years ending

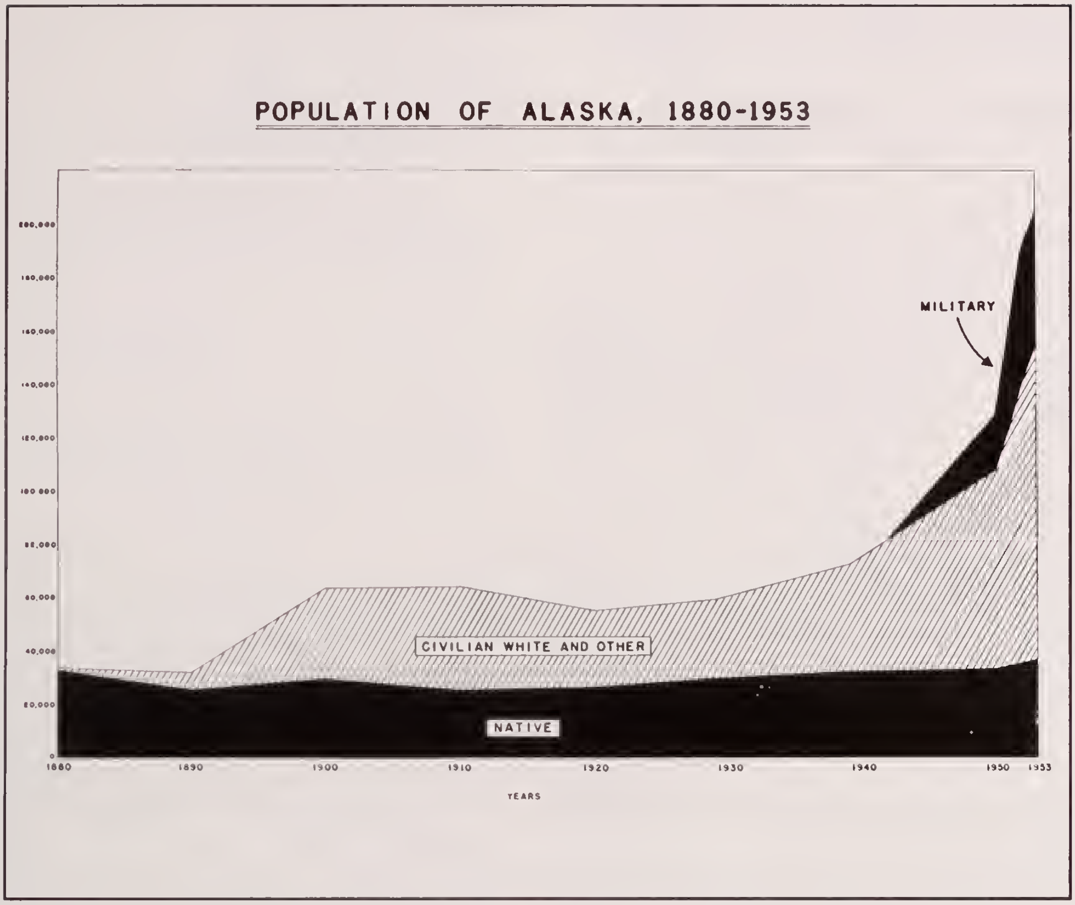
Disease	June 30, 1954	June 30, 1953	June 30, 1952	June 30, 1951
Chicken Pox	435	523	414	585
Conjunctivitis	303	134	136	103
Diarrhea	13	0	12	212
Diphtheria	1	120	2	0
Fish Tapeworm Infection	1	0	0	0
Encephalitis	0	0	1	2
Gastro-enteritis	0	57	7	18
German Measles	61	176	341	97
Gonorrhea	252	298	743	539
Impetigo	288	159	40	8
Infectious Hepatitis	84	5	384	28
Influenza	614	1422	888	559
Measles	3183	159	1039	802
Meningitis	0	6	10	6
Mononucleosis	2	1	5	1
Mumps	1005	397	209	177
Pertussis	1	132	87	80
Pneumonia	20	49	24	37
Poliomyelitis	98	129	25	73
Rheumatic Fever	2	3	5	21
Ringworm	20	2	34	7
Roseola	11	20	0	0
Scabies	2	16	22	29
Scarlet Fever	23	9	43	17
Strep Sore Throat	96	55	69	40
Syphilis	14	40	132	108
Trichinosis	7	0	0	0
Tuberculosis	1072	540	525	350
Typhoid Fever	6	5	1	6
Undulant Fever	0	3	0	0
Vincent's Angina	2	0	1	8
Weil's disease	0	4	5	0


DEATH RATES BY AGE AND RACE, STATES AND ALASKA 1950.

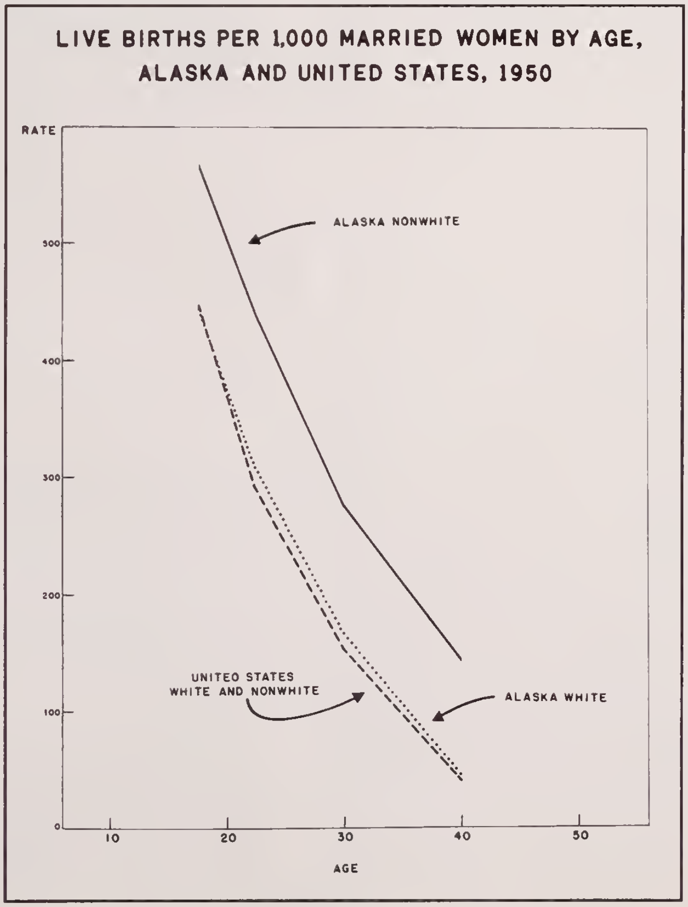
DEATHS FROM SELECTED CAUSES
AMONG ALASKA WHITES AND NATIVES AND STATES WHITES 1950.

DEATHS PER 100,000 PERSONS



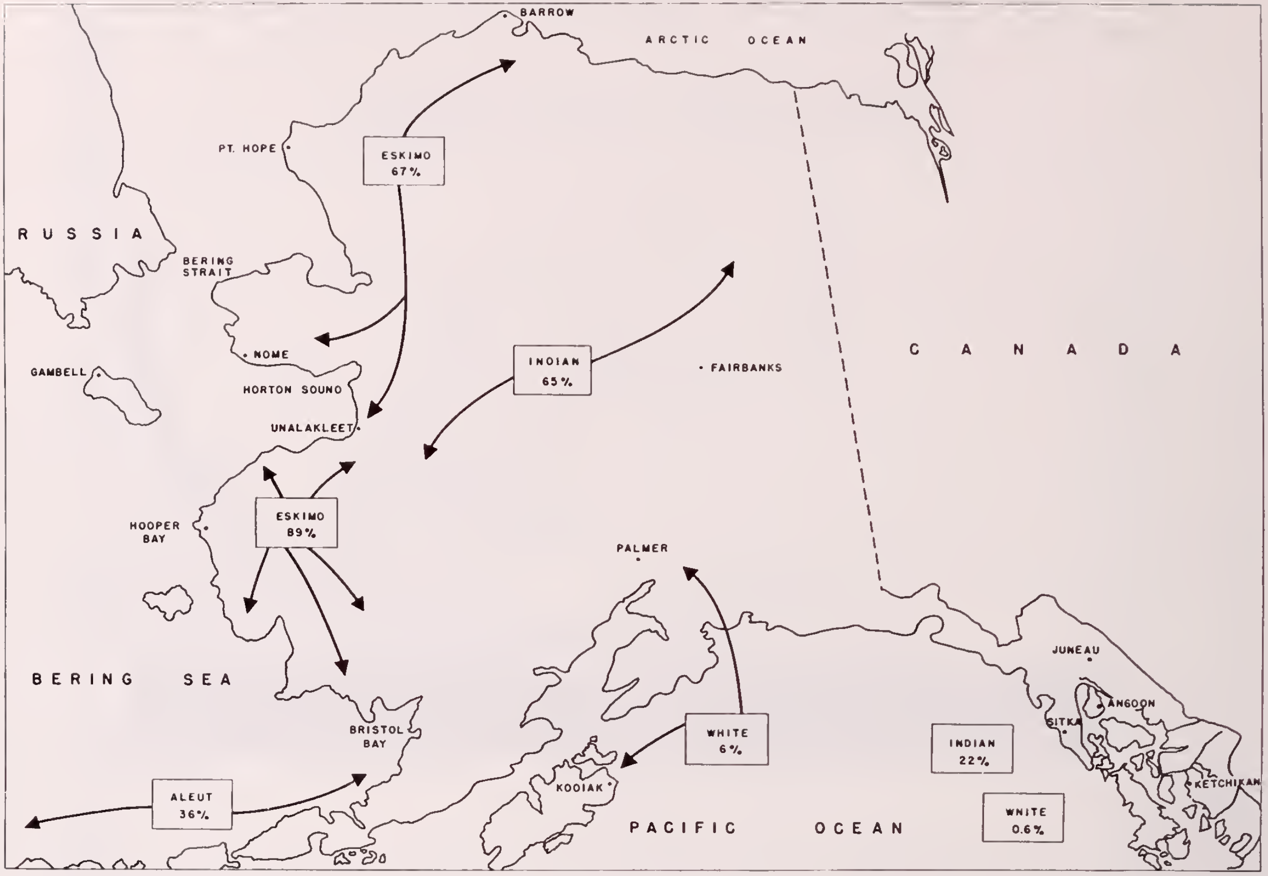


POPULATION OF ALASKA, 1880-1953



LIVE BIRTHS PER 1,000 MARRIED WOMEN BY AGE, ALASKA AND UNITED STATES, 1950

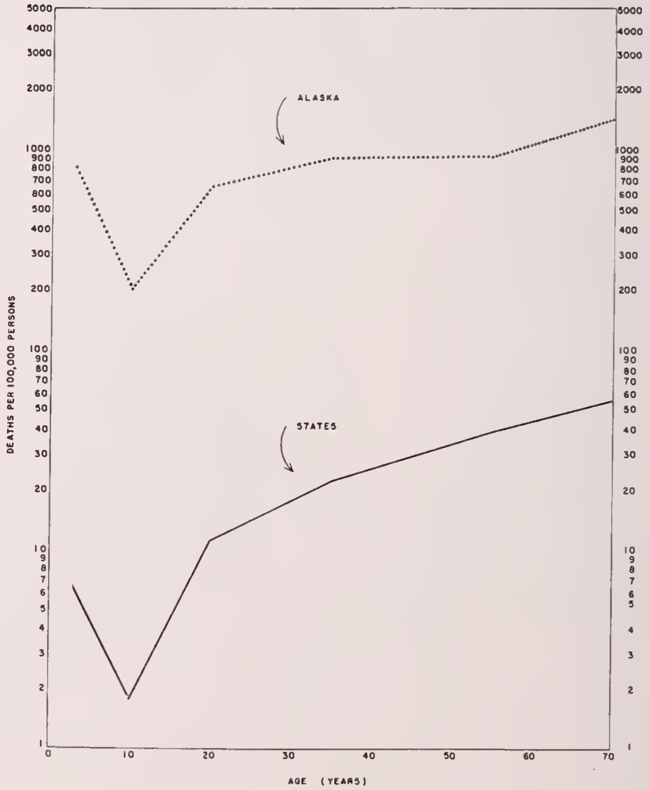
PERCENTAGE POSITIVE TUBERCULIN REACTORS AMONG 5—AND 8—YEAR OLD CHILDREN. ALASKA, 1948-1951



Residents of Alaska Hospitalized for Tuberculosis

Hospitals	Patients
Anchorage	275
Firland Sanitarium, States	4
Hudson Stuck Memorial	10
Juneau Alaska Native Service	23
Kanakanak	23
Laurel Beach Sanitarium, States	8
Marine, States	8
Maynard McDougall	6
Mental-Morningside, States	15
Mt. Edgecumbe Sanitarium	249
Riverton Sanitarium, States	13
Seward Sanitarium	154
Tacoma Indian Hospital, States	62
Tanana	14
Veterans Hospital, States	7
Miscellaneous, States	4
Total	898

TUBERCULOSIS DEATH RATE BY AGE, ALASKA NATIVES AND UNITED STATES TOTAL POPULATION, 1950



(continued from page 70 - Prevention of Ovarian Cancer)

Nationwide, post simple hysterectomy women are left at risk to develop ovarian cancer. It is estimated that 1 in 70 will do so.

Since bilateral oophorectomy is, at this time, the only definitive way to prevent ovarian carcinoma, we believe oophorectomies should be carried out at time of hysterectomy.

ACKNOWLEDGMENTS

Pam Weiler, R.N. and LuAnn Webb, R.N., BSN, MSN, for their assistance in statistical analysis and data collection.

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Letters to the Editor

ACQUIRED CORNEAL ASIGMATISM

Dear Editor,

In Volume 37, No. 3 Dr. Arnold et al reported on the case of a 13-year-old boy with acquired corneal asigmatism. They must be congratulated for the excellent work-up on this patient and the number of eye care professionals they were able to include in this study. I found the clinical photographs (Figures 1 through 4) and the visual acuities very interesting and the interpretations of these findings worthy of discussion.

The clinical photos show eyes that apear orthophoric and if there is a ptosis present it does not show on these photos. The best corrected vision of the RE is 20/60 with glasses and 20/40⁻ with a contact lens. The LE is of course a 20/20 eye and we can conclude that the vision with both eyes is 20/20. An important finding which was not mentioned was the degree of binocularity with glasses vs contact lenses!

In the discussion section the authors refer to this case as "potentially disabling". With one eye corrected to 20/40 to 20/60 and the other to 20/20 one cannot make a case for a potential disability. The visual status is rmarkably stable in this patient and indeed will likely remain so unless of course the astigmatism worsens to a degree requiring surgery. Since we do not know the status of the astigmatism over the past few years we can only speculate on the basis of the corneal topographic test. Indeed the test does not indicate a tendancy toward progressive disease!

Visual acuity with a spectacle is 20/60 and with a contact lens $20/40^{-}$ is $20/50^{+}$! If one uses this later notation it is apparant that the contact lens offers a one line improvement of vision. To say that this "one liner" would enable the patient to function with "normal" vision and without fear of blindness should he lose the better eye is misleading. Since the eye in question is amblyopic it is highly likely that the patient is unaware of the central visual acuity in that eye let alone whether it is 20/40- or 20/60! This fact also makes it unlikely that this 13-year-old patient will be using the contact lens after one year's time! To ascribe 'sight-saving" attributes to the contact lens implies once again a progressive corneal dystrophy which is controlled by the lens!

The clinical photos do not show a vertical muscle imbalance nor a ptosis but the text describes an inconsistant vertical deviation due to poor fixation. Such a variable muscle imbalance is not unexpected with an amblyopic eye and is not a problem requiring surgical intervention! The authors are to be commended for sharing this case with the readers.

Peter E. Cannava, MD

Obituary

DR. MIKE FRANKLIN

Dr. Mike Franklin died March 1, 1996, at his home in Juneau after a lengthy illness. He was 49.

At his request, no services were planned, but he wished to express his gratitude to his patients and friends who had stood by him over the years, his family said.

Dr. Franklin was born in Seattle on Aug. 4, 1946. He received degrees from the University of Washington and George Washington Medical School. After an internship at Virginia Mason Hospital in Seattle, he began a medical practice in Juneau in August, 1973.

Despite receiving an advanced cancer diagnosis in January, 1977, Dr. Franklin was able to pursue his medical career and devote time to his special interests, which included coaching youth baseball, boating and fly fishing, his family wrote.

He served on the Bartlett Memorial Hospital Board for several years, served as president of the State Chapter of the American Cancer Society, was president of the Gastineau Channel Little League and organized the Juneau Baseball and Softball Association.

Dr. Franklin was responsible for the creation of Alaska's first high school baseball program at Juneau-Douglas High School and more recently organized Juneau's American Legion Baseball Program. He also played in several Gold Medal Tournaments in the late 1970s and early 1980s, and was nominated to several all-tournament teams.

Over the years, Dr. Franklin coached Parks and Recreation youth basketball, Little League and American Legion teams. His family, however, remained foremost in the priorities of life his family wrote.

Survivors include his wife Beatrice; son, Adam; and mother, Vivian. His father, Forrest preceded him in death.

From the Juneau Empire, dated March 1, 1996.

PHYSICIAN, ACTIVIST LOSES HIS BATTLE WITH CANCER

Mike Franklin was known for his tenacity when it came to getting things done, both in the medical profession and out. The well-known Juneau doctor and avid sports fan died Friday after a lengthy bout with cancer.

Franklin was committed to everything he laid his hands on, friends say.

His involvement extended from fighting disease in his medical practice to legislative debates and contests on the fields of Juneau's ballparks, all the while fighting his own battle. In 1977, Franklin was diagnosed as having colon cancer.

"He has been a real inspiration to the community, especially to those of us in the medical community," said Marilyn Freymueller, a Bartlett Memorial Hospital Board of Directors member. "He was a good doctor and a good friend."

In 1973, Franklin began a medical practice in Juneau, along with Dr. James Ratcliffe and Dr. Kim Smith, at Bartlett Memorial Hospital. After Ratcliffe left the practice, Franklin and Smith teamed up with Dr. William Cole and Dr. Eric Olsen, to form Family Practice Physicians.

During the construction of the Family Practice Physicians facility, he was right there said Olsen, "working with the banks, architects and builders to get it all done."

"He was the one of us that had the best business sense," said Olsen. "We'll miss him for the leadership role he showed us."

Diana Kuhns agrees. The executive vice president of the American Cancer Society in Alaska said, "his exemplary leadership was unbelievable. His commitment to the American Cancer Society was incredible."

In 1991 the organization presented him with an Outstanding Project Award for his work in pushing a bill through the 1991 Legislature. That legislation became law, which now requires insurance companies to pay for mammogram screenings under policies which pay for mastectomies, a common breast cancer surgical procedure. The bill became effective in September, 1991. "It was unprecedented," said Kuhns.

Franklin became the vice president of the American Cancer Society for Alaska in 1988 and served until 1990. He became the statewide president in 1990. From 1992 up until his heath, he was one of two nominated state delegates to the organization's national board of directors.

His involvement went well beyond the office walls of medical practice. To Juneau baseball and softball enthusiasts, he remains an unsung hero. "He really started high school baseball in the state of Alaska, and brought back American Legion ball," said Little League organizer Don Cary.

It all started when Franklin's son, Adam, wanted to play organized baseball, but couldn't because Juneau didn't have any teams — not at the high school level or even Little League, said Cary. Franklin soon shipped out a proposal to the Juneau School district. They accepted.

That seed planted by Franklin had a mushrooming effect: IN 1990 the Juneau Baseball and Softball Association was formed; in 1991, Juneau-Douglas High School had its first baseball team; and in 1993 American Legion Baseball was brought back to life by the doctor.

"On a very clear mid-May (1991) evening, we had the very first high school baseball game in the state," said Cary. "There must have been a 1,000 people in Melvin Park."

Franklin's tenacity in providing his son an opportunity to play baseball paid off. His concern is understandable. He had played Division One baseball for the University of Washington; now Adam plays for a Division One program at the University of Portland, Ore., Cary said.

By Chris Russ, the Jueanu Empire, dated March 3, 1996.

(continued from page 64- *Tribal Based Cancer*)

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Book Review

The Medicine of Laughter

by Nicholas Deely, M.D.

Dr. Nick Deely has produced a delightful little book of practice anecdotes which he accumulated during 36 years of pediatric residence and practice throughout Alaska. Many of the 92 "chapters" are quotes from the miniature humans he has dealt with and consist of their charmingly naive view of things. Others are devoted to surprising statements, misconceptions, and misinterpretations of their parents. A few chronicle Nick's adventures during his Alaskan travels conducting clinics, etc.

The book furnishes a pleasant evening's entertainment and I heartily recommend it.

Donald R. Rogers, M.D.
Editor

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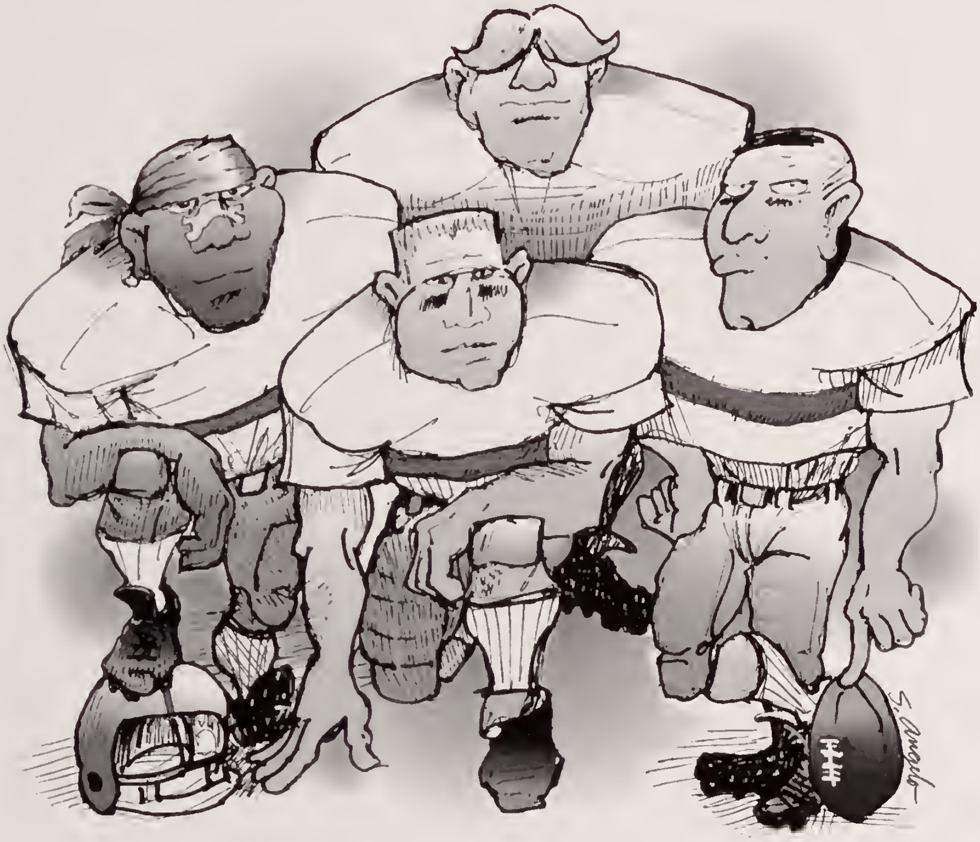

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About the cover: Tanaga Island — Aleutian Islands
Tanaga has three volcanoes - this is the easternmost.

NEONATAL RESPIRATORY DISTRESS CAUSED BY CRYPTIC DACRYOCELES

Robert W. Arnold, MD(1)
Jerome List, DDS, MD(2)
Carl E. Rosen, MD(3)
Eric H. Johnson, MD(4)
Robert O. Hoffman, MD(5)

ABSTRACT

Two cases of neonatal respiratory distress responded with treatment of bilateral dacryocele. In one case, no visible or palpable external evidence of dacryocele was present until mucopurulent discharge commenced more than a week after birth. While the incidence of typical congenital nasolacrimal duct obstruction is about 6%, dacryocele is 200 times less common.

INTRODUCTION

Six to ten percent of normal newborns manifest an obstruction of the distal nasolacrimal duct. A much smaller percent may develop a blue-domed-cyst or dacryocele caused by obstruction of the distal and both proximal valves. Monocular dacryocele has been reported to cause respiratory distress (1). We report a case of neonatal respiratory distress caused by bilateral, externally hidden dacryoceles and a case with respiratory distress and visible dacryoceles..We also attempt to approximate the incidence of dacryocele.

CASE REPORTS

Case 1: A 35-week, large-for-gestational-age caucasian infant was admitted to Providence Hospital NICU for respiratory distress. The pregnancy was unremarkable other than pregnancy-induced hypertension for which a ceasarean section was performed. Apgars were 7 at 1 minute and 8 at 5 minutes. She then had decreased respiratory effort with grunting, flaring and intercostal retractions. A nasogastric tube could not be passed through the right naris. Supplemental oxygen produced pink integument. No râles were heard. A computed tomogram was performed for presumed choanal atresia. No bony abnormalities were noted but bilateral cysts protruded from the lateral nasal walls and the lacrimal sacs (Figure 1). There was no tearing or evidence of orbital masses. A dilated eye exam showed a normal fundus without coloboma. The nasal masses were initially treated with Neosynephrine drops. By the second hospital day, she was weaned to room air. Because of the possibility of obstruction and a family history of apnea, she was dismissed on an apnea monitor. The respiratory difficulty manifested primarily during feeding. On the 12th day of life, a mucopurulent discharge appeared in the otherwise quiet right eye. No dacryocele was visible externally (Figure 2). The remainder of her

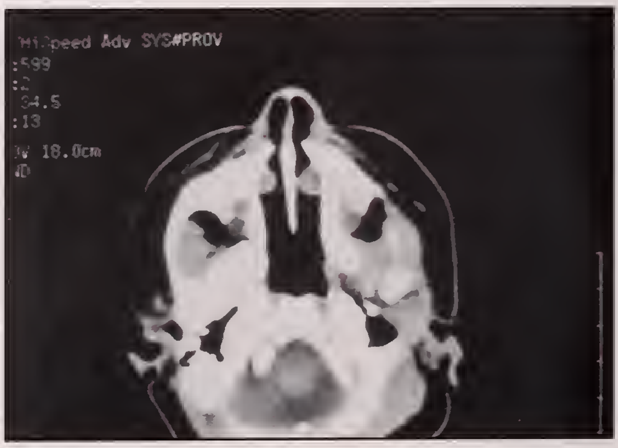

Figure 1. Computed tomogram of case 1, a neonate with respiratory distress and failure of nasogastric tube passage through the naris. Note the bilateral dilated nasolacrimal sacs partially obstructing the nasal airway.

---

(1) Pediatric Ophthalmology and Strabismus
(3) Orbit, Plastic and Neuroophthalmology
Ophthalmic Associates, a P.C., 542 West Second Avenue, Anchorage, Alaska 99501-2242.
(2) ENT; Head and Neck Surgery, 3300 Providence #208, Anchorage, Alaska 99508.
(4) Alaska Neonatology Associates, 3340 Providence Drive, Ste 366, Anchorage, Alaska 99508.
(5) Pediatric Ophthalmology, University of Utah, 50 North Medical Drive, Salt Lake City, Utah 84132.

eye exam remained normal, specifically there was no evidence of keratitis, uveitis or congenital glaucoma. Using a 000 Bowman probe, each nasolacrimal system was probed in the office. There was a reflux of mucopurulent material from the right side and amber fluid from the left. Obstructions were encountered at the valves of Rosenmüller of each inferior canaliculus and at the Valve of Hasner bilaterally. The lumen of the nasolacrimal sac was unusually large. Care was taken to avoid aspiration. A steroid-antibiotic drop was prescribed. Respiratory symptoms cleared.

One year later, no epiphora persisted but the child had multiple episodes of otitis media which were moderately responsive to oral antibiotics.

Case 2: An urgent ophthalmology consult was sought for a 14-hour-old hispanic female infant. She was the product of a full term, uncomplicated pregnancy. Respiratroy distress was noted with feeding or supine positioning with oxygen desaturations below 85%. Examination (by R.O.H) showed bluish appearing subcutaneous lesions, left larger than right, inferior to the medial canthal tendon bilaterally. Ultrasound confirmed continuity of boeth of these cystic lesions with the ipsilateral nasolacrimal duct.

The patient was taken to the operating room where, under general anesthesia, nasolacrimal duct probing was performed bilaterally. Intranasal examination showed large cystic lesions below the inferior turbinate bilaterally. These were shown to be part of the lacrimal drainage system by palpating a 00 Bowman probe through the cyst wall when passed from above. An opening was then cut in the intranasal cyst under direct visualization with a V-Lance blade with immediate decompression of the lacrimal system noted. The opening was enlarged until flourescein-stained saline, irrigated from above, passed without significant resistance or recurrance of the cyst. Post-operatively, topical antibiotic/corticosteroid drops were used in conjunction with lacrimal sac massage for 5 days. The cystic lesions had not recurred at the one week follow-up examination.

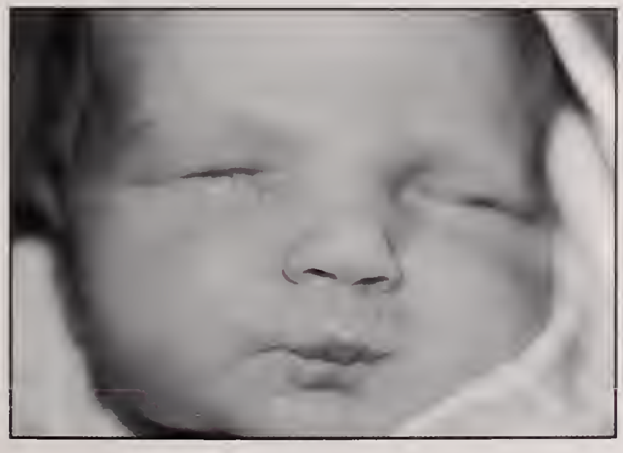

Figure 2. Twelve-day-old infant with persistent respiratory difficulty, one day of right eye mucopurulent discharge and no evidence of blue-domed cyst.

DISCUSSION

Since neonates are obligate nasal breathers, obstruction of the nasal passageways will produce respiratory distress which usually improves during crying. In addition to dacryocele, the differential diagnosis for upper airway, neonatal respiratory distress includes choanal atresia, foreign body/inspissated nasal secretions, congenital cysts (dermoid, mucous retention, saccular cyst of the larynx), encephalocele, Thornwalt bursa, craniopharyngioma, trauma, syphilis, and edema (2,3).

We present two cases of dacryocele, an entitiy which requires more urgent treatment than typical congenital nasolacrimal duct obstruction. More than 5% of infants, and especially those with persistent otitis media, have an obstruction of a Valve of Hasner at the distal end of the nasolacrimal sac (4). The onset of mucopurulent discharge is from weeks to months after birth. Spontaneous resolution with occasional topical antibiotics administered concurrently with gentle massage-mixing occurs in 80-90% of cases by 7 months or 15 pounds. Younger infants (less than 6 months) may be treated with non-sedated, restrained nasolacrimal probing in an outpatient setting. Prolonged obstruction is often treated with probing under general anesthesia. Failed probings, or recurrent symptoms which occur in 10-30% of probings especially in older children, are usually treated with silicone stents. This "gold-standard" stenting usually requires an office visit for removal although the Crawford tube can be safely and reliably removed at home if installed by the "Farson-modification" (5). Resolution of nasolacrimal duct symptoms is about 98% in the absence of allergic rhinitis by this technique.

Dacryocele, (also known as congenital nasolacrimal mucocele (6), or amniotocele) is much rarer because it involves obstruction of both Rosenmüller Valves in addition to the Valve of Hasner. Dacryoceles present as a bluish raised rubbery mass inferior to the medial canthus. Although initially sterile, they will frequently cause an acute dacryocystitis with potential for orbital cellulitis. Unilateral dacryoceles can cause respiratory obstruction when the bulbous distal portion fills a naris (1). Although some resolve with conservative therapy (7), the treatment of choice of typical dacryocele is probing. From 6/90 through 12/93, one of us (RWA) cared for 7 dacryoceles (3 OD, 3 OS, 1 OU) compared to 141 surgical corrections for persistent nasolacrimal duct obstruction in children 6 months to 14 years. This translates to one case of dacryocele in 2-3000 births. There is a strong association between otitis media and conventional nasolacrimal duct obstruction (Sprunger, DT and Morse C, AAPOS abstract 1994).

Dacryocele must be considered in the differential diagnosis of neonatal respiratory distress even in the absence of a visible or palpable blue-domed cyst. Imaging studies may speed treatment in cryptic cases.

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INCREASE IN rT_3 SERUM LEVELS OBSERVED DURING EXTENDED ALASKAN FIELD OPERATIONS OF NAVAL PERSONNEL

Percival D. McCormack, MD, PhD(1)
Harrell L. Reed, MD(2)
John R. Thomas, PhD(1)
Munnazza J. Malik, MS(1)

ABSTRACT

Members of a US Navy Special Warfare platoon had blood samples drawn by venipuncture and other baseline measurements carried out prior to departure for a three-month period of field operations in Alaska. Assays of serum reverse triiodothyronine (rT_3), free thyroxine (fT3), free T_4(fT_4), and thyroid stimulating hormone (TSH) were subsequently done at the Naval Medical Research Institute (NMRI), Bethesda, Maryland. In the field and at baseline, hematocrits and urine specific gravities were also measured to track hydration status, and body weights and fat were recorded to track nutritional status. After plasma volume change and weight change corrections, an approximate 30% increase in serum rT_3 level and 20% decrease in free T_3 level over 76 days of cold exposure were recorded. The necessity for an rT_3 kinetic study is indicated. The mean residence time for rT_3 in the circulation is about 8 hours with respect to about 36 hours for free T_3, so rT_3 kinetic studies would be advantageous with respect to long term cold exposure of military personnel.

INTRODUCTION

Evidence has been accumulating for some time that human physiology adapts to changing environmental factors such as gravity (31,28), time zone (19), altitude (32) and temperature (1,11,25). In the context of environmental stress the thyroid is a major reactive system, so it is appropriate to briefly review thyroid metabolism. Thyroxine, the major secretory product of the thyroid gland, is metabolized by the peripheral tissues by phenolic conjugation, deamination, decarboxylation, and a cascade of monodeiodinations. One product, 3,5',3' - triiodothyronine (T_3) is the major active form of the thyroid hormone, and about 80% of the T_3 produced in the body is derived extrathyroidally. Moreover, a greater fraction of the T_3 found on nuclear receptors in pituitary and brain cells is derived intracellularly as compared with liver and kidney cells. The latter tissues, on the other hand, appear to be the source of most of the circulating T_3. This is achieved by outer ring phenolic deiodination of thyroxine (T_4), regulated by the enzyme 5'-deiodinase Type I (3). Another deiodinase, acting on the non-phenolic (tyrosyl) ring of T_4, produces the (apparently) hormonally inactive 3,3',5'- triiodothyronine (reverse T_3 or rT_3). A number of physiological and pathological events perturb the deiodination pattern, leading to a decrease in T_3 neogenesis and reciprocal changes in the circulation of T_3 (which decreases) and rT_3 (which increases). This is called the "low T_3 syndrome." The biological effects resulting from these changes are, at present, incompletely understood, but are potentially important in the body's adjustment to stress.

The extrinsic influence of cold exposure on iodothyronine homeostasis in a group of Navy personnel during a 3-month operational exercise in Alaska is the focus of the present paper. The primary variable studied was the rT_3 serum level. A recent study (2) has reported reciprocal changes in serum T_3 and rT_3 in subjects exposed to high altitude and cold over a period of two weeks. There was a rise in rT_3 and fall in T_3. Other cold studies have not included the hormone rT_3 and will be reviewed here. It has been found (8) that circulating iodothyronines and thyrotropin do not alter significantly in adults during a 30-minute to two-hour cold challenge when not previously exposed to cold (cold naive). When human subjects underwent multiple (-20 to 8°C) or extended (12°C) air exposure (22, 26) for 12 to 14 days, they showed changes in both thyroid hormone kinetics and static serum levels. In one cold study (21) involving 16 men exposed to cold air (4°C) for 30 minutes, twice a day for eight weeks, the T_3 Plasma Clearance Rate (PCR) and T_3 Plasma Appearance Rate (PAR) increased by about 18% after fourteen days of the protocol and remained elevated over the next six weeks of the cold exposure.

---

(1) Thermal Stress Division (Code 53), Naval Medical Research Institute, 8901 Wisconsin Avenue, Bethesda, Maryland 20889-5607.

(2) Department of Medicine, Madigan Army Medical Center, Tacoma, Washington 98431, USA

Reed et al. (21) have studied subjects during extended residence in Antarctica, with only hands and face exposed to the cold outdoor temperatures. Energy intake was determined to increase by only about 40% without a change in body weight with respect to pre-deployment measurements (4). A decline in both serum free T_3 (fT_3) and body temperature was weakly correlated after extended residence. Moreover, serum Thyroid Stimulating Hormone or thyrotropin (TSH) response to Thyroid Releasing Hormone (TRH) was augmented by about 50% (23). An approximate 17% decline in the T_4 Volume distribution (Vd) without an increase in T_4 PAR was also observed. These changes in static measures were associated with a more than doubling in T_3 Vd, PAR and PCR (24). Another more recent study confirmed a small decrease in fT_4 (about 6%) and Total T_4 (tT_4)- about 8% during 10 serial monthly measurements in Antarctica (12). These findings were interpreted as a primary change in T_3 kinetics and distribution, involving a replacement of about 17% of the T_4 pool with T_3 and thus doubling the T_3 pool. This has been called the "polar T_3 syndrome."

It also has been reported that military personnel operating in circumpolar locations, who are involved in strenuous physical exercise with marginal nutritional intake and partial sleep deprivation, have declines in tT_3 and tT_4 regardless of their housing conditions(22,16). The fT_3 and fT_4 are increased after 5 to 10 days and decreased after 60 days. T_3 PCR and Vd are increased and TSH is insufficient after 60 days to return fT_4 to pre-deployment levels. It appears that the various influences of undernutrition, sleep deprivation and exercise do not inhibit either the T_3 kinetic changes of cold exposure (22), or the development of cold adaptation (14). In view of the reciprocity in serum T_3 and rT_3 levels which appears in various forms of stress - surgical trauma (9,17,27) and hyperthermia (13) - sometimes referred to as the rT_3/T_3 syndrome - it would be expected that serum rT_3 levels would rise during long term cold exposure (30 days or more). It must be noted that rT_3 kinetic changes are an extremely sensitive indicator of undernutrition, with the activity of 5'-deiodinase Type I decreasing significantly with mild (15%) undernutrition and a measurable decrease in rT_3 PCR noted during simple energy restriction (15).

The present study involved the measurement of circulating rT_3 levels in 16 healthy males from a US Navy Special Warfare platoon during a 3-month period on a winter training exercise in Alaska.

MATERIALS AND METHODS

The medical records of all test subjects were reviewed with respect to nutritional and cardiopulmonary problems. Baseline data on each subject was taken (at Little Creek, Virginia) just prior to deployment to Alaska. This included the following:

(i) routine physical examination
(ii) body composition: nude dry weight, height and the sum of skin-folds at 7 sites (thigh, abdomen, etc.). Body surface area, body surface to mass ratio, body fat % and lean body mass were calculated from this data(7).
(iii) hydration status: determined from urine samples tested for specific gravity, and from a 5 ml venous blood sample quantified for hemoglobin and hematocrit, and used to calculate blood and plasma volume shifts.
(iv) orthostatic tolerance: assessed by the stand test and used to quantify orthostatic tolerance as another indicator of hydration status(5).
(v) 10 ml of blood by venipuncture, used to measure baseline levels of rT_3, fT_3, fT_4 and TSH.

When filled, blood containing test tubes were immediately placed in an ice container and within a few hours were spun down, the serum pipetted off and transferred to EDTA lined tubes. These were then stored in dry ice for transportation to the Naval Medical Research Institute (NMRI) analytical laboratory in Bethesda, Maryland. Long-term storage was in freezers at a temperature of -70° C. Assays of fT_3, fT_3 and TSH were carried out by DPC RIA kits designed for the quantitative measurement of these hormone plasma levels. rT_3 assays were carried out using RIA kits from Wien Laboratories Inc of New Jersey.

Mean baseline weight of the 16 subjects was 78.7 kgm; mean age was 25.63 years; mean body fat was 6.29%.

Field Studies in Alaska

Observations were carried out on all test subjects on five separate occasions over a period of 76 days. This covered 14 days at Little Creek, Virginia and 62 days in Alaska. The same measurements and interventions (venipuncture) were carried out as listed above for the baseline studies. Taking the baseline as zero days, the studies were at plus 35, plus 38, plus 55, and plus 76 days. This study was part of a larger study assessing the potential benefits of tyrosine administration in the cold. The

Table 1. Experimental Plan for Field Studies

Time	Measurements and Blood Samples
+ 35 Days	Before Tyrosine Dose and One Hour Cold Exposure
	After Tyrosine Dose and One Hour Cold Exposure
+ 38 Days	Before Cold Exposure and Exercise (5K Ski Run and Target Shoot - 3 Hours)
	After Cold Exposure and Exercise (5K Ski Run and Target Shoot - 3 Hours)
+ 55 Days	Before Cold Exposure and Target Shoot - 6 Hours)
	After Cold Exposure and Target Shoot - 6 Hours)
+ 76 Days	Before Tyrosine Dose and One Hour Cold Exposure
	After Tyrosine Dose and One Hour Cold Exposure

experimental plan is outlined in Table I.

The project protocol was approved by the Human Use Committee at the NMRI. The study itself was carried out in accordance with the Helsinki convention. During these cold exposures the mean ambient daily temperature over the 76 days was -3.9° C (minimum temperature -6.7 and maximum -2.4° C).

The test subjects regularly performed outdoor activities including physical exercises, running, skiing, maneuvers, rifle shooting, etc. They had frequent and daily exposure to the cold environment, often with exposed hands and faces. There were approximately 18 hours of darkness each day over the 76 days. Nights were spent largely indoors. Caloric intake never fell below 4,200 per day for all subjects.

Tyrosine/rT_3 Interaction in Blood: The question of alteration in measured serum rT_3 levels due to the presence of tyrosine was addressed. A standard solution containing 500 ng/dL of rT_3 was diluted with zero standard (0 ng/dL of rT_3) and then further diluted with pooled plasma. The concentration was measured at each dilution to verify the dilution factor. A stock solution was next prepared of tyrosine in pool plasma (540 nmol/ml) - this was equivalent to the dose of tyrosine given to the test subjects in Alaska. A further dilution was made in the standard to bring the concentration of rT_3 to 175 ng/dL. 100 microliters of the stock solution was added to 350 microliters of this solution (tyrosine plus pool plasma) to bring the total volume to 450 microliters. Five standard/stock solutions were assayed, with the tyrosine component varying from 75 to 5 microliters, and the rT_3 concentrations were measured. The results will be presented in the next section.

Correction for Plasma Volume Changes (6): Because the measured changes in plasma hormone concentration might be partly due to the effects of hemoconcentration or hemodilution (hydration status), the measured changes were corrected according to the equation:

Corrected Concentration = Measured Concentration (100 +% D PV)/100 ....(1)
where DPV = change in plasma volume with respect to the baseline value obtained prior to deployment.

The results will be presented in the next section.

Correction for Weight Change: Because of the sensitivity of rT_3 serum levels to nutritional status, it was necessary to estimate the effect observed weight changes would cause. Data from Goode et al. (10) was used to estimate the changes in rT_3 levels due to weight variation alone. The correction factors used were as follows:

+4.75% change in rT_3/ 1% loss of weight
-4.75%change in rT_3/ 1% gain in weight.

For T_3 values the correction constant was +/- 3.9, from the data of Goode et al. (10). The positive sign applies in this case (T_3) to gain of weight. The results of the rT_3, freeT_3, free T_4, and TSH assays will be presented in the next section.

Statistics:

The Windows program Statistica (version 4.5) was used for all statistical analyses of the experimental data. Normal probability plots and the Shapiro-Wilks test were used on each set of data (uncorrected or raw data and weight and plasma volume corrected, at each of the 5 observation times) to establish that the distributions were normal. To identify outliers and extreme data values, Box Plots were constructed, again for the uncorrected and corrected values. The effect of outliers was minimized by replacing them by the mean value (30). ANOVA was then used to produce plots of the means. Finally, scatter plots of the means and linear regression graphs were produced for the uncorrected and corrected sets of data. A table of means and standard error of the means was also constructed.

RESULTS

Tyrosine/rT_3 Interaction: Table II shows data on the measured rT_3 plasma levels (in pg/ml) as the added tyrosine concentration is varied over a range of 15. A regression analysis of plasma rT_3 level versus tyrosine level was carried out. The correlation coefficient is -.751, but the p-value is .14; therefore, the correlation is not significant. The results indicate an association and so it

Table II. **Tyrosine/rT_3 Interaction**

Standard	Tyrosine	Pool Plasma	rT_3 pg/ml
350 µl	75 µl	25 µl	160.3
350 µl	50 µl	50 µl	171.47
350 µl	25 µl	75 µl	173.5
350 µl	10 µl	90 µl	170.86
350 µl	5 µl	95 µl	196.67

Table III. **Mean rT_3 Serum Levels (pg/ml) Versus Time (days)**

Time	0	35	38	55	76
Uncorrected rT_3	27.4±1.04	28.38±1.08	28.63±1.64	32.54±1.76	34.28±2.31
(PV + Weight) Corrected rT_3	27.4±1.04	29.79±1.15	30.2±1.54	33.19±1.84	34.90±2.16

Table IV. **Free T_3, Free T_4, and TSH (pg/ml) Versus Time (days)**

Time	0	35	38	55	76
T_3 Uncorrected	2.82±.19	1.83±.14	1.99±16	2.21±18	2.22±.19
T_3 Corrected	2.82±.19	2.10±.13	2.09±16	2.38±.14	2.14±.13
T_4 Corrected	1.07±.04	1.06±.04	1.87±.04	1.09±.05	1.00±.04
TSH Corrected	1.52±.11	1.77±.12	1.84±.12	1.28±.14	2.37±.16

was decided to assay plasma rT_3 levels in blood samples taken before the oral administration of tyrosine.

rT_3 Data Analysis: As described in the last section the raw data was first examined statistically to establish that the rT_3 values at each time of observation were distributed normally across the 16 test subjects. The Normal Probability Plot of the baseline measurements and a Shapiro-Wilks value of $p < .5265$ established that the data were indeed distributed normally. This was repeated for each of the other 4 observation times and in each case similar results were obtained - all demonstrated normal distributions. Table III shows the means (+/- standard errors) of the measured and uncorrected serum rT_3 values versus time of exposure in Alaska.

Using ANOVA a plot of the uncorrected means versus time was obtained and is shown in Fig. 1. The analysis indicated that the difference in rT_3 values between time 0 (baseline) and time 4 (76 days) was significant. The scatter plot of rT_3 versus time and the linear regression graph which has a correlation coefficient of +.91 is shown in Fig. 2. The p value for the linear regression was .054 - just outside the accepted limit for statistical significance. The linear regression line would predict a change of (30+/-10)% in the mean rT_3 serum levels over the 76 days of observation.

Corrected rT_3 data: The raw rT_3 data was next corrected for plasma volume and weight variation (7) according to eqtn.2.

rT_3 corrected = rT_3 uncorrected (1 + PVC) + WC*4.75 ....(2)

where PVC is the Plasma Volume Change Fraction

WC is the Weight Change Fraction

The corrected rT_3 values were checked for normal distribution by Normal Probability Plots. All distributions were normal. The final corrected rT_3, mean rT_3 values and SEMs are also shown in Table III. Fig. 3 shows the Scatter Plot of corrected rT_3 values and linear regression graph. The correlation coefficient was .997 and the p was <.006 - highly significant. A (29.4+/-9.5)% change in rT_3 serum values is indicated over the 76 day exposure.

Free T_3 Data Analysis: The data analysis was done in the same way as for the rT_3 analysis. Normal Probability Plots and Shapiro-Wilks p values showed that the data at each observation time had normal distributions (Shapiro-Wilks $p<.21$).

Table IV lists the adjusted raw mean fT_3 values and SEMs versus time. The ANOVA analysis indicated that the difference between the mean fT_3 values at the first two points (zero time and 35 days) was significant. Fig. 4 is the scatter plot of the uncorrected mean free T_3 values versus time and the linear correlation graph. The linear correlation coefficient is only -.53 and $p<.51$ (indicating poor linear correlation and not statistically significant).

Corrected fT_3 Data: The raw fT_3 data were corrected for plasma volume and weight changes according to eqtn. 3.

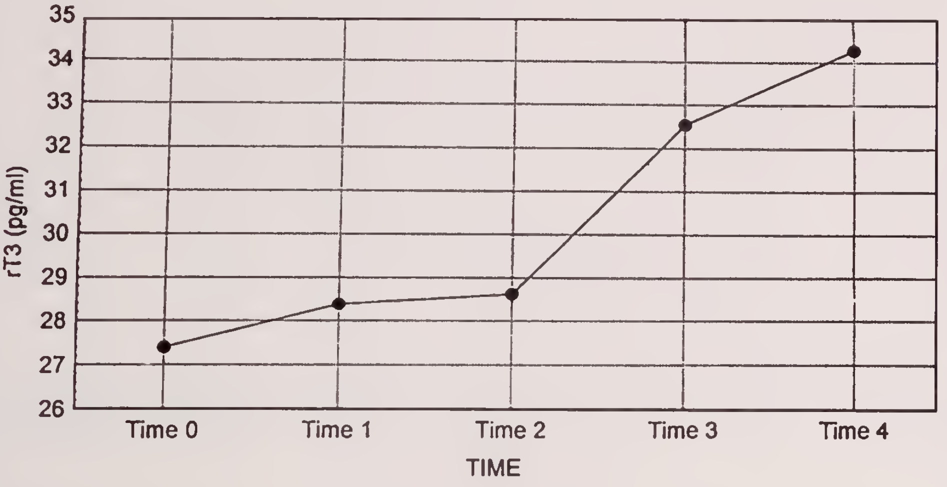

Fig. 1. Uncorrected mean serum rT_3 concentrations versus time of observation. rT_3 concentration in pg/ml. (Time 0 is baseline; Time 1 at 35 days etc).

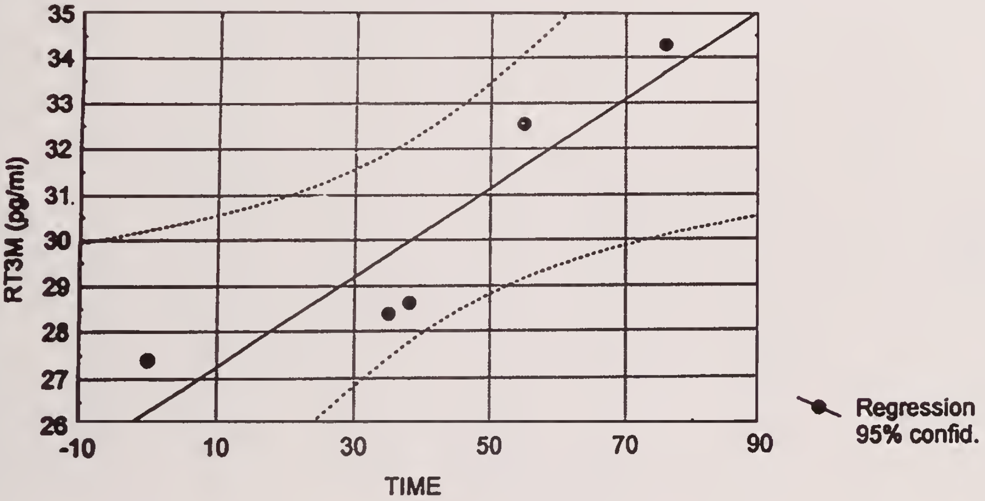

Fig. 2. Scatter plot and linear regression graph of uncorrected mean serum rT_3 concentrations versus time. rT_3 concentrations in pg/ml and time in days.

fT_3 corrected $= fT_3$ uncorrected $(1+PVC) + WC*3.9$ ....(3)

The corrected data were analyzed as before and the corrected mean fT_3 values and SEMs are presented in Table IV. Fig. 5 shows the scatter plot of Corrected Mean Values of fT_3 versus time and the linear regression graph. The correlation coefficient is now -.67 with a p value of .22. Again, there is a linear correlation of fT_3 serum concentration with time, but it is not statistically significant. The linear fitted graph would predict a decrease in the corrected mean fT_3 values of (20+/-8)% over the 76-day period. It is apparent that a linear relation is not appropriate for this data. Some form of polynomial fit would be more accurate.

Free T_4 and TSH Data: The data for both of these hormones at each observation time were normally distributed. Correction for plasma volume changes was made. The corrected mean free T_4 and TSH values plus SEMs are shown in Table IV. The scatter plots and linear regression graphs are presented in Fig. 6 (fT_4) and Fig. 7 (TSH). The linear regression line for fT_4 has a slope of virtually zero and indicates that there is no significant correlation of fT_4 and time of cold exposure. The linear correlation line for TSH has a correlation coefficient of

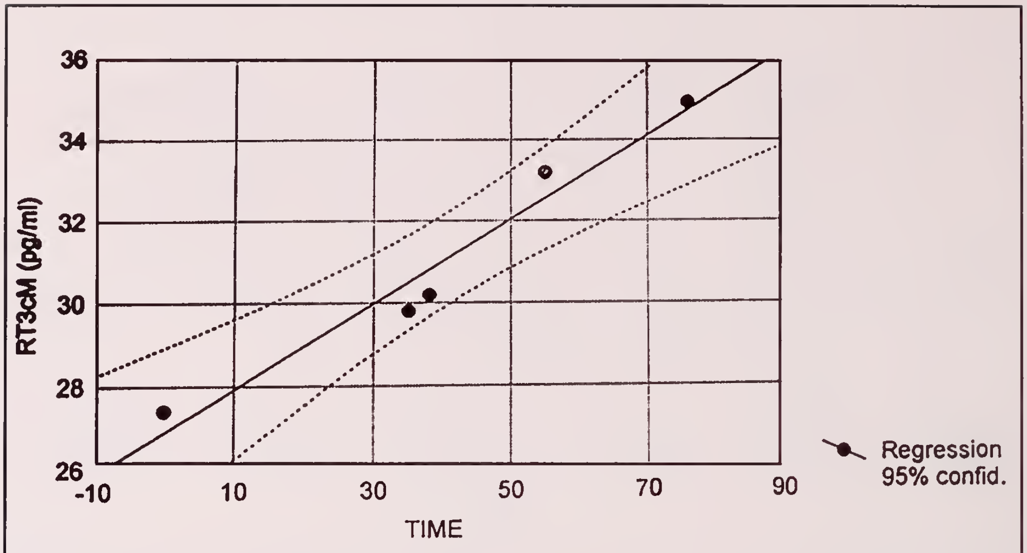

Fig. 3. Scatter plot and linear regression graph of corrected mean serum rT_3 concentrations versus time. rT_3 concentrations in pg/ml and time in days.

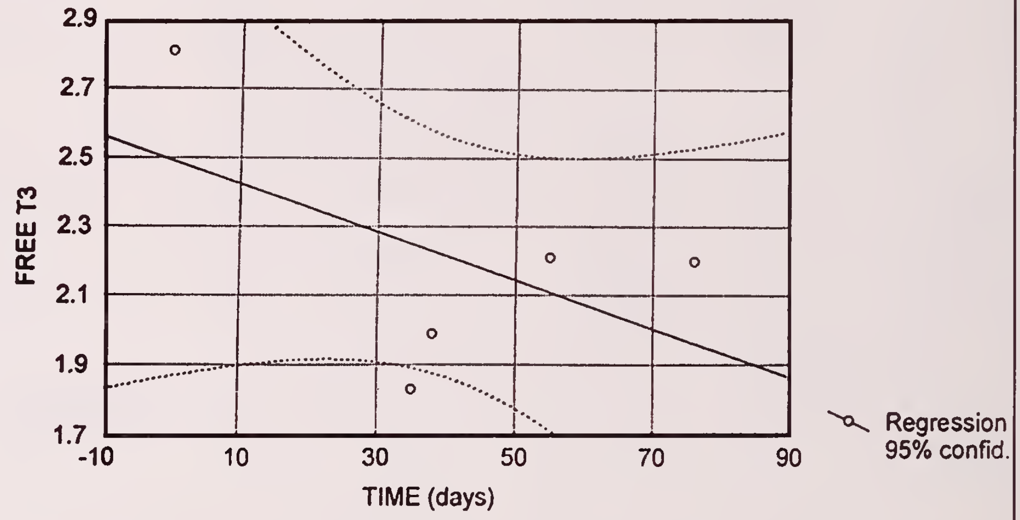

Fig. 4. Scatter plot and linear regression graph of uncorrected mean serum free T_3 concentrations versus time. Free T_3 concentrations in pg/ml and time in days.

.53 and a p value of .36, indicating a weak correlation which is not statistically significant. The linear graph predicts an increase in serum TSH level of (34 +/- 14)% over the 76 days period.

DISCUSSION

The environments near the geographic poles are marked by prolonged periods of darkness in winter and extended winters with cold temperatures. Adaptive responses of serum thyroid hormones have already received considerable attention, and in this paper these observations have been extended by measurement of the serum rT_3 response to long-term cold exposure. A summary of the main results is as follows:

(i) a (29.4 +/-9.5)% rise in serum rT_3 level - statistically significant.
(ii) a (20 +/-8)% fall in serum free T_3 level - not statistically significant.
(iii) no change in serum free T_4 level.
(iv) a (34 +/-14)% rise in serum TSH level - not statistically significant.

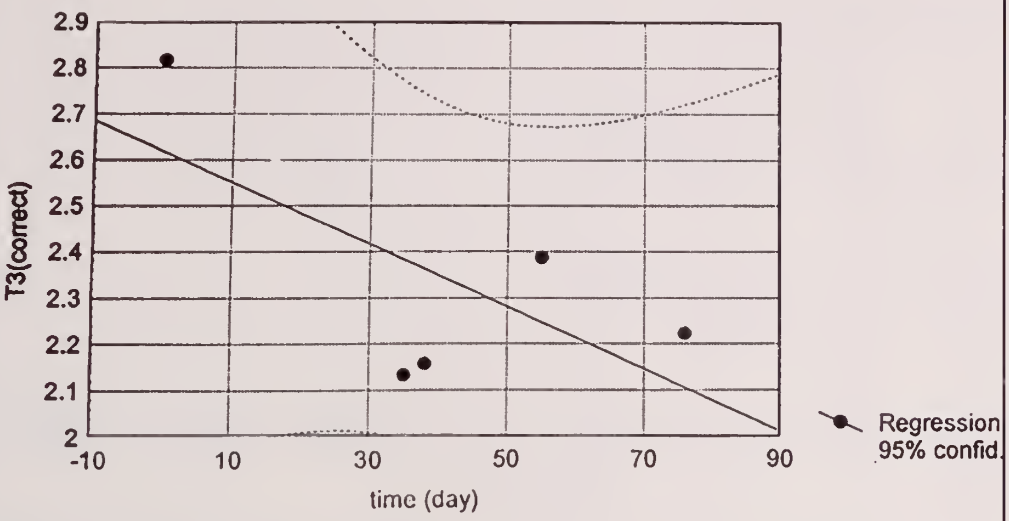

Fig. 5. Scatter plot and linear regression graph of corrected mean serum free T_3 concentrations versus time. Free T_3 concentrations in pg/ml and time in days.

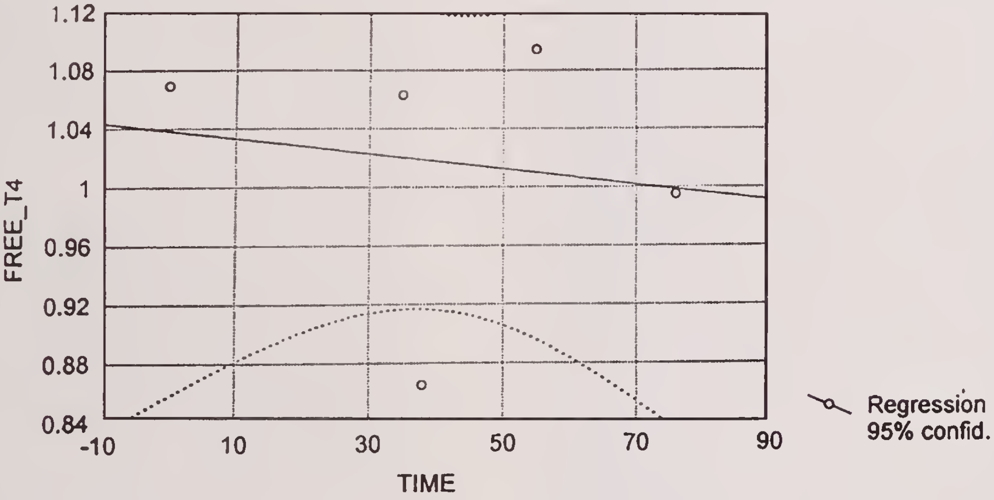

Fig. 6. Scatter plot and linear regression graph of corrected mean serum free T_4 concentrations versus time. Free T_4 concentrations in pg/ml and time in days.

The results for free T_3, free T_4, and TSH are supported, qualitatively at least, by those reported by Reed et al. (20) for extended Arctic residence. The reciprocity of changes in free T_3 (falls with cold exposure) and rT_3 (rises with cold exposure) has been observed to occur with other stressors - surgical trauma (9,17,27) and hyperthermia (13).

Circulating thyroid hormones represent only a small fraction of the total body pool for thyroxine (T_4 -28%), triiodothyronine (T_3) - the active thyroid hormone (14%) - and reverse T_3, the apparently inactive hormone mediator (14%). Therefore, changes in the kinetics of these hormones are more revealing than simply measuring circulating values (19 and 30). Reed et al. (22) have recently developed and used a non-radiolabelled T_3 kinetic methodology during cold air exposure and Antarctic field operations. Use of this technique allowed multiple studies over an 8-week course of cold air exposure and field studies located in isolated areas. It can easily be extended to rT_3 kinetic studies. There is the added advantage that rT_3 is the preferred substrate for 5'-deiodinase Type I, and its mean residence time is about

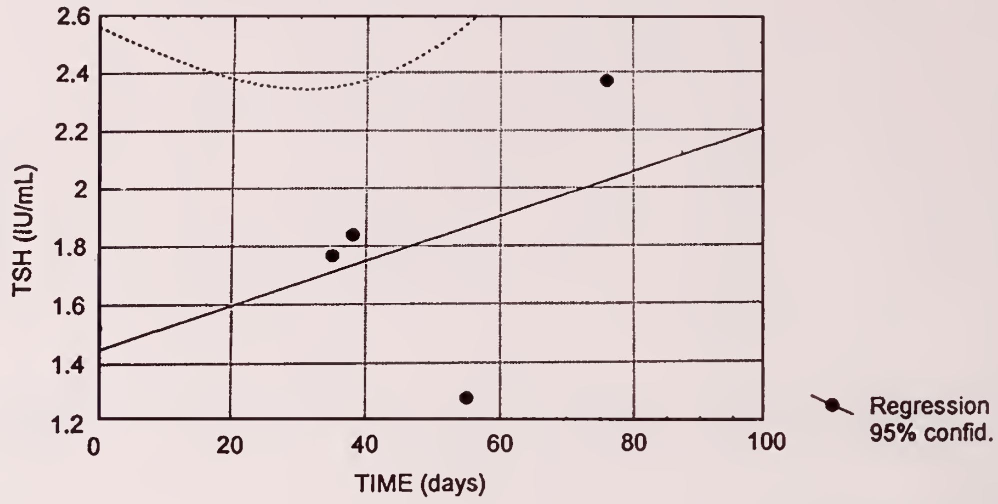

Fig. 7. Scatter plot and linear regression graph of corrected mean serum TSH concentrations versus time. TSH concentrations in IU/ml and time in days.

8 hours compared with about 36 hours for T_3. Thus kinetic studies with rT_3 are nearly 4 times shorter - an important consideration for personnel operating in the field. As mentioned previously, rT_3 kinetic changes are an extremely sensitive indicator of undernutrition, with the activity of 5'-deiodinase Type I declining significantly with mild (15%) undernutrition and a measurable decrease in rT_3 PCR noted during simple energy restriction (29). The results reported in this paper suggest that a similar decrease in 5'-deiodinase Type I activity occurs with long-term cold exposure. Therefore, rT_3 kinetics studied with a non-labelled hormone could provide meaningful information in the operational theater and could be carried out in a safe and reproducible manner. If rT_3 kinetics is used as a sensitive in vivo tool to measure the major enzymatic regulating enzyme, 5'-deiodinase Type I, a better understanding could result of environmental settings where the effects of stressors such as hypoxia, exercise, undernutrition, cold exposure, and hypergravity combine to alter thyroid hormone economy with subsequent performance changes.

ACKNOWLEDGMENTS

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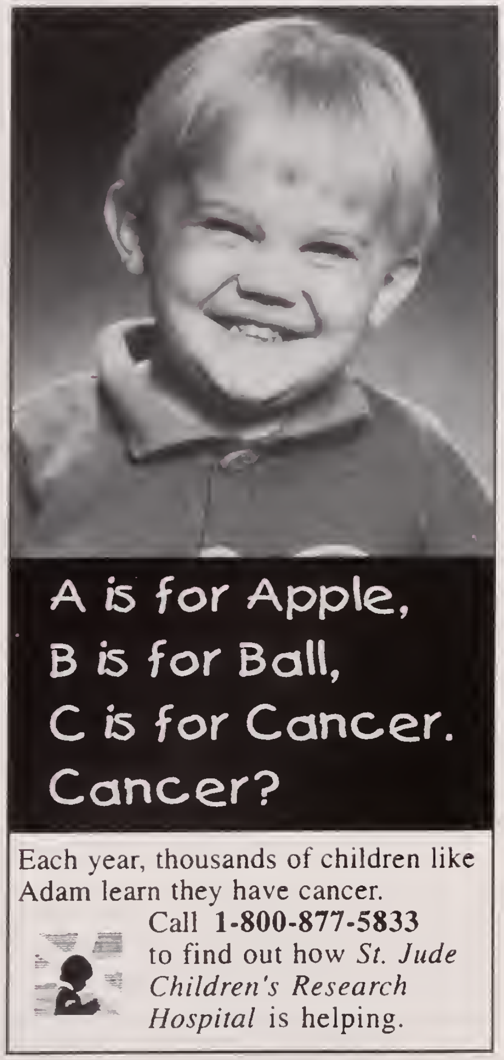
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BILIRUBINEMIA IN ASPHYXIATED TERM INFANTS

A CLINICAL OBSERVATION

Harry Harrison, Jr, MD(1)
Ronald W. Coen, MD(2)

ABSTRACT

Recent clinical observations suggested that serum bilirubin levels remain low following perinatal asphyxia. This study was performed to determine the peak serum bilirubin concentrations in asphyxiated term infants and to compare these levels with those in a group of non-asphyxiated term infants. The medical records of 28 asphyxiated (Apgar <7 at 5 minutes) and 947 non-asphyxiated term infants born during the same period of time were reviewed to obtain data on peak serum bilirubin concentrations for statistical comparison. The statistical mean for the peak serum bilirubin concentrations in the 28 asphyxiated infants was 5.0 mg/dl (+3.1 SD) while that for the 947 non-asphyxiated infants was 8.9 mg/dl (+3.7, p <.001). The findings in this study indicate that low peak bilirubin levels should be expected in asphyxiated term infants and may be helpful as a biochemical marker before the mechanism of action is known.

INTRODUCTION

Serum bilirubin concentrations in the newborn infant should rise following perinatal asphyxia as the result of hypoxic-ischemic injury to the liver, including hepatic necrosis (1), and the presence of closed-space tissue hemorrhages. Recent clinical observations suggested that serum bilirubin levels remain low following perinatal asphyxia This study was performed to determine the peak serum bilirubin concentrations in asphyxiated term infants and to compare these levels with those in a group of non-asphyxiated term infants.

MATERIALS AND METHODS

The medical records of 28 asphyxiated (Apgar <7 at five minutes) and 947 non-asphyxiated (Apgar 2 7 at five minutes) infants born during the same period of time were reviewed to obtain data on peak serum bilirubin concentrations for statistical comparison. Term gestation was determined by the Dubowitz scoring system and defined as gestational ages between 38 and 42 weeks. The infant's hospital course and those factors which relate specifically to bilirubin metabolism were noted from the records.

The 28 asphyxiated infants (female =14, male =14) were managed in a newborn intensive care facility. Eighteen (64%) required mechanical ventilation and intravascular plasma volume expanders (e.g. 5% albumin). Intravenous fluids (50 cc/kg/d) were used in all 28 cases during the first two days and none received enteral feedings during the first forty-eight hours of life. The presence of skin bruises and hematomas were noted. Liver function studies were available in 13 (46%) of the asphyxiated infants. Twenty-two (79%) asphyxiated infants passed meconium during parturition.

One asphyxiated infant was treated with phototherapy when the serum bilirubin peaked at 11.2 mg/dl; however, another infant with the highest peak bilirubin (12mg/dl) was not treated with phototherapy. A major blood group (ABO system) incompatibility with a positive direct Coombs test existed in one other asphyxiated infant. Three infants developed a disseminated intravascular coagulopathy (DIC), but did not undergo exchange transfusions. Phenobarbital was administered to 16 (57%) asphyxiated infants to treat seizures. Results of bilirubin levels after hospital discharge were not used in this study.

All non-asphyxiated infants received routine care in a newborn nursery. Twelve percent (112/947) of the infants passed meconium during parturition, and 28 of the 112 (25%) were intubated in the delivery room to remove meconium from the airway. Despite the early airway manipulation these infants had uneventful clinical courses in the nursery. Bilirubin levels were not investigated for any of the infants in the non-asphyxiated group after hospital discharge.

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(1) Alaska Neonatology Associates, 3340 Providence Dr., #366, Anchorage, Alaska 99508.
(2) Pediatrics/H638A, University of California Medical Center, 225 Dickinson Street, San Diego, California 92103.

Student t-test or chi-square was used to compare mean values for the peak serum bilirubin concentrations in the asphyxiated and non-asphyxiated infants. Further analyses were made to determine if either the presence of meconium at birth, treatment with phenobarbital, or abnormal liver function tests related significantly to observed differences. Mean values for gestational age and admission weight were also compared.

RESULTS

The mean admission weight for the asphyxiated infants was 3544 grams (_552 am) and mean admission weight for the non-asphyxiated group was 3610 grams (+490 am). The difference in mean admission weight was not significant (p =NS). There was a difference in gender distribution between the two groups, but this was not significant (p <.1). The mean gestational age for the asphyxiated and non-asphyxiated infants was similar (40.4+1.2; and 40.1+1.1 weeks respectively; p =NS).

Within the group of asphyxiated infants there was no evidence that meconium passage during parturition, administration of phenobarbital, hepatic injury, or disseminated intravascular coagulation made a significant difference in the mean peak bilirubin level (Table 1). Therefore, the full group of 28 asphyxiated infants was compared to the non-asphyxiated group of infants.

The mean peak serum bilirubin concentrations in the 28 asphyxiated term infants was 5.0 mg/dl (+3.1 SD) while that for the 947 non-asphyxiated infants was 8.9 mg/dl (+3.7, p <.001).

Mean peak bilirubin concentration was lower (5.4 mg/dl) in 16 asphyxiated infants treated with phenobarbital than in the non-asphyxiated infants (p <.01). Six (21%) asphyxiated infants suffered hepatic injury as evidenced by markedly elevated serum levels of liver enzymes. However, the mean peak bilirubin concentration (6.1mg/dl) was significantly lower than that of the non-asphyxiated group of infants (p <.01).

The peak bilirubin concentration in three asphyxiated infants with DIC did not exceed 6.8 mg/dl. One infant with a blood group incompatibility had a peak bilirubin of 5.2 mg/dl. These values were lower than the mean peak bilirubin concentration of the non-asphyxiated group and in the same range as the remainder of the asphyxiated group.

DISCUSSION

This study indicates that the group of asphyxiated and non-asphyxiated term infants were comparable for gestational age and admission weight. Fekete et al (2) found that perinatal asphyxia did not exaggerate hyperbilirubinemia in term infants. The effect of early meconium passage and phenobarbital administration was not mentioned. Mulligan et al (3) and Paxson (4) both commented on the lack of hyperbilirubinemia in asphyxiated infants. However, neither author quantitated serum bilirubin levels or noted clinical factors which influence these levels. Phenobarbital administration has been shown to be a useful adjunct in the treatment of neonatal hyperbilirubinemia, but studies have not compared non-asphyxiated and asphyxiated infants receiving this drug (5).

Although this series of asphyxiated infants is small, the data highly suggest that meconium passage during parturition and phenobarbital administration do not explain the lower serum bilirubin levels in asphyxiated term infants. Phototherapy is not a factor since it was used on only one asphyxiated infant in this series. In addition, two of the asphyxiated infants died on days 6 and 8, so there was sufficient time for bilirubin levels to rise in all 28 infants; yet they did not.

The findings in this retrospective study indicate that

Table 1.

Asphyxiated Term Infants (n=28)

	Unmatched Comparison		*Bilirubin in mg/dl		p value
Phenobarbital	[16+]	12-	[5.4]	4.5	(P>.4)
Meconium at Parturition	[22+]	6-	[5.5]	3.4	(P>.1)
Hepatic Damage	[6+]	22-	[6.1]	4.8	(p>.3)
DIC	[3+]	25-	[5.6]	5.0	(p>.7)

* Mean Peak Bilirubin Concentration

(DIC) disseminated intravascular coagulation
[ ] represents same group data

low peak bilirubin levels should be expected in asphyxiated term infants, and may be helpful as a biochemical marker before the mechanism of action is known. The factor(s) responsible for this finding are unknown. Further studies of heme oxygenase inhibition and induction of heme oxygenase mRNA transcription following asphyxia might yield an explanation (6,7).

ACKNOWLEDGEMENT

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APOLOGIA

In the spring issue of Alaska Medicine which was devoted to tobacco issues, we published an article entitled "Tobacco Control and the AMA" by Dr. Thomas P. Houston, the director of the Department of Preventive Medicine and Public Health of the AMA. The article was a composite of two that he had previously published in the medical journals of the states of North Carolina and Indiana. This was accomplished by Dr. Anne Lanier and approved prior to publication.

We failed to attribute the original articles from which the distillation was prepared to those journals. We apologize.

1. Houston TP. The role of organized medicine and the American Medical Association in tobacco control. NC Med J 1995;56:20-22.
2. Houston, TP. Tobacco control and the AMA: Health, policy and politics. Indiana Med 1996;89:129-131

Donald R. Rogers, MD
Editor

The Evaluation and Treatment of Congestive Heart Failure in Alaska

Mary Ellen Gordian, MD, MPH[1]

INTRODUCTION

The prevalence of congestive heart failure (CHF) rises exponentially with increasing age (1). CHF is the greatest single cause for hospitalization of Medicare patients in Alaska and, in addition almost 40% of these patients have multiple admissions within a year. Improving the treatment of CHF could substantially effect morbidity and mortality and reduce hospital costs. The purpose of this study was to review the care of patients with CHF in Alaska. The study was done by abstracting all charts of patients identified from Medicare claims data as having been hospitalized during the given time period with the primary diagnosis of CHF.

The Agency for Health Care Policy and Research (AHCPR) and the American College of Cardiology have recommended guidelines for diagnostic work-up and treatment of CHF based on the results of clinical trials and in concurrence with expert opinion (2). We chose two indicators from the guidelines for which there was consensus and evidence of efficacy. We wanted to look at both the diagnostic process and the treatment of CHF. The two indicators are 1) a documentation of an evaluation of left ventricular function, and 2) the use of angiotensin converting enzyme (ACE) inhibitors at hospital discharge.

BACKGROUND

CHF can be defined as a clinical syndrome of dyspnea and fatigue caused by left ventricular dysfunction. Heart failure due to the inability of the left ventricle to adequately empty itself is called systolic dysfunction; whereas, diastolic dysfunction is the inability of the left ventricle to adequately fill (3). Systolic and diastolic dysfunction have different pathophysiologies and require different therapies. The distinction between systolic and diastolic heart failure is clinically important as some therapies that may be helpful for systolic dysfunction can be deleterious to patients with diastolic dysfunction. Heart failure can be the result of intrinsic problems with the heart itself or the result of systemic problems (anemia, hyperthyroidism, etc). In most cases echocardiography can not only determine the left ventricular ejection fraction, but it also can provide information about treatable causes of dysfunction.

Left ventricular systolic dysfunction is the most common cause of heart failure; however, if the ejection fraction is greater than 40% diastolic dysfunction should be considered (4). Measurement of left ventricular performance is a critical step in the evaluation and management of all patients with suspected or clinically apparent heart failure. The combined use of history, physical examination, chest x-ray, and EKG is not sufficient in determining whether a patient's symptoms and physical findings are due to dilated cardiomyopathy, left-ventricular diastolic dysfunction, valvular heart disease, ischemic LV dysfunction or a noncardiac etiology. Echocardiology or radionuclide ventriculography can substantially improve diagnostic accuracy (5).

One of the effects of the reduced cardiac output associated with heart failure is the release of renin which initiates a series of enzymatic reactions resulting in the conversion of angiotensin I to angiotensin II. This increases both pre-load and after-load with increased systemic vascular resistance, and sets up a vicious cycle causing further deterioration of cardiac function (6). ACE inhibitors interrupt this cycle and achieve a number of beneficial hemodynamic effects. While Captopril, a short-acting ACE inhibitor, and enalapril and lisinopril with longer duration of action (up to 36 hours), have been the ACE inhibitors most studied in the treatment of heart failure, other ACE inhibitors already in use as antihypertension medications are expected to have the same effects in heart failure. Losartan which has a different mechanism of action has not been tested in clinical trials for this indication.

ACE inhibitors comprise the only therapy that has been shown in randomized controlled trials to reduce mortality and readmissions for congestive heart failure (7). ACE inhibitors have also shown dramatic improvements in clinical, hemodynamic, and exercise variables. While the cost of ACE inhibitors is higher than conventional therapies, the additional efficacy of this treatment makes the cost per year of life saved much lower than that of other accepted medical therapies (8).

METHODS

The study population was composed of Medicare beneficiaries 65 years of ago and older who were admitted to Alaska hospitals between October 1, 1993 and

September 30, 1994 with a primary diagnosis of congestive heart failure(ICD-9-CM of 402.01, 402.11, 402.91, 404.01, 404.03, 404.11, 404.13, 404.91, 404.93, 425.4, 428.0, 428.1, 428.9, 429.3, 518.4). Only hospitals which had 30 or more CHF admissions in the study year were invited to participate. A total of 290 patients were identified from seven hospitals. If multiple admissions occurred for a patient during the study period, the last admission was selected for inclusion in the study. Patients with missing charts (three) or an incorrect ICD-9-CM code (ten) were excluded from chart abstraction. Of the 277 cases selected for chart abstraction, 14 were excluded because of in-hospital death, and one for unknown discharge status.

The chart abstraction tool was designed to collect information from inpatient medical records including patient demographics and processes of care. The abstraction tool was alpha and beta tested by PRO-West to attain a 95 percent agreement rate. A ten percent random sample was evaluated for inter-rater reliability. Overall agreement for the data used was 90 percent. We not only looked at the performance rate of the two indicators, we also looked at the number of tests that were given to patients with the diagnosis of CHF.

DEFINITIONS

Evaluation of Left Ventricular Function

Documentation of left ventricular ejection fraction was present if it was noted that an echocardiogram, ventriculography, radionuclide ventriculogram, MUGA, or any mention of an ejection fraction measurement was made in the patient history; or if any of these procedures were done during the admission.

LVEF measurements were considered either moderately to severely decreased, or normal to slightly decreased based on the following definition:

- moderately to severely decreased LVEF- if there was a record in the chart of either LVEF < 40 percent or a written description of a moderately or severely decreased LVEF.
- normal to mildly decreased LVEF if there was a record in the chart of either LVEF 40 percent or a written description of a normal or mildly decreased LVEF.

ACE inhibitors

The list of ACE inhibitors includes benazepril, captopril, enalapril, fosinopril, lisinopril, quinapril, ramipril, and any combination of these with diuretic. If any of these were listed by generic or Trade names as discharge medication, then ACE inhibitor at discharge was considered present.

Contraindications used in this study were taken from AHCPR guidelines. If a patient had one or more of these contraindications recorded in their chart, they were considered to have a contraindication to ACE inhibitor use, even though we recognize that these contraindications are not absolute:

- history of renal artery stenosis
- past history of an adverse reaction to an ACE inhibitor.
- serum creatinine levels greater than 3.0 mg/dL
- serum potassium levels greater than 5.5 mEq/L

Cardiologists

Where the attending physician was listed as a cardiologist in the Alaska State Medical Society directory, the patient was coded as having been cared for by a cardiologist, otherwise, they were cared for by non-cardiologist. No attempt was made to distinguish between family practice and internal medicine specialists.

RESULTS

Table 1 shows the demographic characteristics of the patients in the study. Table 2 lists the average number of tests that were done during the hospitalization. This is stratified by urban and rural hospitals and by cardiologist and noncardiologist as attending physicians.

Table 1.

Demographics of Medicare patients with CHF

	Urban	Rural
Age 65-74	59	61
Age 75+	75	68
Male	59	58
Female	75	71
Median LOS*	5.3	3.5

*an average of the median LOS for each hospital

Fifty-three percent of all CHF patients (N=263) had documentation of having had an LVEF measurement done either prior to or during admission (see Table 3). The rate at different hospitals varied from 8% to 80%. The usual clinical procedure for obtaining a measurement of the ejection fraction was an echocardiogram (92%), although eight patients had radionuclide ventriculogram or left ventriculography done either prior to or during admission. There were 165 clinical procedures done on 139 individual patients to measure ejection fraction. Twenty-six patients had a measurement noted in the history and an additional procedure done during admission. Twenty-three of these were repeat echocardiograms. Of the patients with no admission for CHF in the last 12 months, 61% had an LVEF measure-

Table 2.

Processes of Care in Congestive Heart Failure - Average number of tests per admission

	BUN	CR	K+	CBC	UA	ABG	X-ray	EKG
Urban hospitals	3.6	3.7	4.6	2.3	1.3	2.0	2.5	2.0
Rural hospitals	3.5	3.6	5.2	2.7	1.2	2.9	2.1	2.6
Cardiologists	3.6	3.6	4.8	1.6	1.3	2.8	2.4	1.9
Noncardiologists	3.6	3.6	4.9	2.7	1.3	2.5	2.2	2.3
All Cases	3.6	3.6	4.9	2.5	1.3	2.5	2.3	2.3

Table 3.

Left Ventricular Ejection Fraction documented in chart (noted in History or done during Admission)

All hospitals	139/263	53%
Rural hospitals	49/129	38%
Urban hospitals	90/134	67%
Cardiologist attending	28/40	70%
Non-cardiologist attending	111/223	50%

ment noted on the chart.

Of the 139 patients who had an LVEF evaluation, 48 of them were noted to have ejection fraction <40%. Fifteen of these patients had ejection fractions of less than 40% documented in their past history and six of them had repeat procedures during the admission. Two of those were noted to have improved ejection fractions on the admission procedure that were above 40%. For purposes of this study, those two patients were retained in the group with systolic left ventricular dysfunction.

There was a significant difference between urban and rural hospitals in obtaining LVEF measurements. The range for three urban hospitals was 60-80% of patients with documented LVEF measurement, whereas at the four rural hospitals, the range was 8- 59%.

The use of ACE inhibitors at discharge was defined as the percentage of those patients without known contraindications who might otherwise be eligible for treatment with ACE inhibitors (see Table 4). Of the 236 patients with no contraindication for the use of ACE inhibitors, only 129 (55%) were discharged on ACE inhibitors. There was no difference between rural and urban hospitals and the range between hospitals was 50% to 67%.

Of those patients for whom ACE inhibitors are specifically recommended by AHCPR guidelines, that is, those with systolic left ventricular dysfunction, there were 48 identified by having an LVEF < 40%. Forty-four of these had no contraindication to ACE inhibitors, but only 26 (59%) of them were discharged on ACE inhibitor (see Table 5).

There were 40 patients cared for by cardiologists. Of these 28 (70%) had an LVEF documented in the chart and 11 were noted to have an ejection fraction less than 40%. Cardiologists used ACE inhibitors in 54% of all the patients without contraindications. Among those with documented systolic dysfunction, however, cardiologists discharged 70% of eligible patients on ACE inhibitors. Non-cardiologists discharged 56% of eligible patients with documented systolic dysfunction on ACE inhibitors.

Best practices for this study were observed at one urban hospital where 78% of CHF patients had documented measurement of left ventricular function and 67% of the eligible patients were treated with ACE inhibitors including 70% of those with systolic dysfunction.

DISCUSSION

The demographic classification of rural and urban patients was similar. Median length of stay was almost two days longer in urban hospitals, but there were no significant differences in the average number of tests done in either location.

This study based on medical record abstractions does not distinguish severity of illness. The indicators were chosen to be as free of bias for severity of illness as possible. Similarly this study is based on aggregate data and does not address the care of individual patients. The optimal rate of each indicator is not known and must be

Table 4.

ACE Inhibitors at Discharge
All CHF patients without contraindications

All hospitals	129 / 236	55%
Rural Hospitals	63 / 116	54%
Urban Hospitals	66 / 120	55%
Cardiologist Attending	19 / 35	54%
Non-cardiologist Attending	110 / 201	55%

Table 5.
ACE Inhibitors at Discharge
Patients with LVEF< 40% and No contraindications to ACE inhibitors

All hospitals	26 / 44	59%
Rural Hospitals	5 / 9	56%
Urban Hospitals	21 / 35	60%
Cardiologist Attending	7 / 10	70%
Non cardiologist Attending	19 / 34	56%

decided locally. The hospital with the best practice and the experience of the cardiologists indicates that 70- 80% of their patients with congestive heart failure have an examination of left ventricular ejection fraction done at some time during their evaluation. Since guidelines recommend that all patients presenting with congestive heart failure have an evaluation of LVEF during their initial workup, then 70-80% compliance may still be less than the optimal level of care, although problems with documentation may account for some of the observed non-compliance.

Best practices in the state for ACE inhibitors in CHF show only 67% of all eligible patients being treated with ACE inhibitors upon discharge. Because diastolic dysfunction may or may not respond to ACE inhibitors, the guidelines established from clinical trials only apply to systolic dysfunction. When we look only at the treatment of systolic dysfunction, we find that the number of patients is very small. Urban hospitals, probably only because they evaluate LVEF more often, have a greater number of patients with systolic dysfunction (N=37). Of those 35 were eligible for treatment, but only 21 (60%) were treated with ACE inhibitors. Our best practice hospital and cardiologists treated 70% of their eligible systolic dysfunction patients with ACE inhibitors.

AHCPR guidelines include hypotension as a contraindication for treatment with an ACE inhibitor. I did not include hypotension as a contraindication. Because of this decision the numbers of eligible patients may be artificially high. Similarly, a few patients that we identified as having contraindications were discharged on ACE inhibitors. Contraindications are relative to each patient's condition and the benefit derived from the medication. The rate of use of ACE inhibitors was based only on patients without contraindications as specified.

Because we abstracted only the last admission for patients with multiple admissions during the year, it was suggested that the left ventricular function was being measured but not mentioned in the history at every admission. To test this hypothesis we looked at 161 patients who had had no previous admission for CHF during the preceding 12 months. In urban hospitals the rate of documentation of left ventricular function increased from 67% to 79% in these patients. In rural hospitals the rate increased from 38% to 43% in this category. The number of new patients presenting with CHF was equal in urban and rural hospitals.

The difference between urban and rural hospitals in documenting LVEF may be explained in part by access to echocardiography. Since the Medicare patients presenting to rural hospitals with CHF are half of all the Medicare patients in the state with the problem, the availability of this technology should not be limited to urban hospitals.

Studies on the effects of using clinical guidelines have shown them to be effective both in improving the processes of care and in improving outcomes.[9] The probability of guidelines being effective are greatly improved if the guidelines are internally developed. Specific educational inventions are more likely to result in improvements than journal articles. The strategy that works best to implement change is patient specific reminders at the time of contact such as protocols that are generated by a given diagnosis and can be signed off or modified by physicians.

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(continued on page 108)

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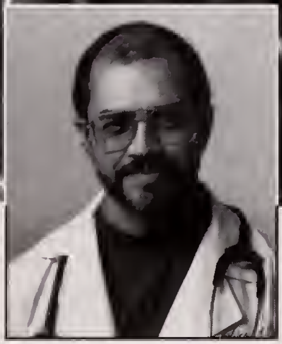

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Malpractice in the New Millennium: Better Than Today?

Managed Care, Telemedicine, Genetic Tests Create New Liabilities

The practice of medicine is at a crossroads. Many physicians speak of early retirement and of discouraging their children from pursuing a medical career. Physicians-in-training, heeding warnings about a surplus of specialsists, are entering primary care residencies in record numbers. Proposals in 1993 and 1994 to transform the nation's health care system fizzled or, as some charge, were euthanized by insurance interests and by politicians worried about a potential loss of campaign contributions. Managed care has emerged as the "new paradigm," enticing physicians and hospitals into fragile, ambivalent relationships. Despite their unrivaled position atop the national pedestal of admiration, many physicians still worry that every patient is a potential litigant. Americans love their doctors and believe that no country has a better health care system. Nevertheless, medical scientists have been productive, and report that a number of extraordinary diagnostic and treatment breakthroughs are within reach. But the excitement about their discoveries is tempered by mounting fears over the long-range legal and ethical consequences of medical research.

Managed care: a work in progress

Managed care clearly is a dominant force American medicine today, and it is likely to remain so into the new millennium. Many health professionals and patients are dubious about managed care. Physicians who decry the disadvantages of managed care medicine nevertheless feel compelled to embrace it, making it their principal source of income. Offered as a proven solution to the nation's health care financing crisis, managed care is largely a work-in-progress. Subscribers and providers sign contracts with some MCOs whose basic policies, procedures, reimbursement schedules, rules, and subscriber information still are being developed. To some observers, managed care is like an airplane that is being constructed during the night.

Supporters assert that managed care results in better and wiser utilization of limited health care resources and dollars. They cite benefits such as shorter hospital stays, reduced use of expensive technologies and fewer referrals to expensive specialists. Capitation, a fixed per-patient fee, is promoted as the best way to central health care costs.

Critics, including some physicians and hospitals already under contract with MCOs, say that managed care is usurping medicine's prerogatives to determine acceptable standards of care, and that what an MCO considers proper utilization and what physicians think is good medicine may not be the same. MCOs speak of "cost-effective medical care," and try earnestly to convince physicians and patients that the term is synonymous with "medically-appropriate care." Some physicians complain that MCOs ask them to provide medical services that exceed their training and experience, which increases their liability risks. For many health professionals, managed care means increased paperwork, additional staff, and re-shaping doctor-patient relationships. Managed care's opponents claim that capitation tempts some physicians and hospitals to withhold necessary care and treatment.

MCOs tout polls that show patients have high praise for managed care. At the same time, reflecting growing public and professional concerns, state legislatures are flooded with proposed laws to regulate managed care. In some states, new laws ban contractual "gag" rules that prohibit physicians from criticizing an MCO or discussing alternative treatment not approved by the managed care organization; in a few states managed care organizations can no longer "deselect" physicians who advocate for patients whose MCOs refuse to pay for recommended medical care. Recently-enacted laws in some states now require MCOs to provide plain-language descriptions of covered benefits, and to establish timely grievance procedures.

Media targets managed care

The media have been savaging managed care. The cover story in *Newsweek's* October 23, 1995, issue was a hit-piece titled, "Beware Your HMO." The cover story in *Time's* January 22, 1996, issue was a "Special Investigation" the magazine called, "What Your Doctor Can't Tell You — An in-depth look at managed care and one woman's fight to survive." *Time's* cover photo is of a physician muzzled by a surgical mask, an allusion to the "speak-no-ill" provisions in some MCO contracts. All of the major TV newsmagazines have aired stories that emphasize the dark side of managed care medicine. A few high profile malpractice cases, such as the one that resulted in an $89 million judgment against Health Net, and other verdicts that juries seem to be using to punish the alleged excesses of managed care organizations and complicit physicians, are among the highest personal injury litigation damage awards ever reported. Some of these cases involved alleged rule-bending, or pressure by MCO higher-ups on physicians to withhold care — always grist for the media — and are likely reasons for the unusually high damage awards. The handful of managed care verdicts in the $20 million to $ 100 million range may be aberrations, but they also may be ominous warnings. Adverse publicity about the alleged excesses of managed care is welcomed by plaintiffs' lawyers, who see malpractice cases that involve injuries to managed care patients as their new cash cow. Attorneys call managed care, "The Lawyers' Full Employment Act of 1996."

Managed care profits draw attention

And then there's the money. Some managed care organizations are becoming giants. Megamergers have given some MCOs extraordinary bargaining power and control over access to health care resources. High administrative margins and bare-bones reimbursements boost profits. Soon, critics say, most medical care in the United States will be controlled by multi-million dollar executives and multi-billion dollar corporations whose domination can systematically force the medical profession to its knees, and send an already-genuflecting Congress into campaign finance heaven. Physicians, journalists, lawmakers and the public are increasingly uncomfortable with the notion that health care economics may become more important than the health care itself. Liability insurers are concerned about the potential for a malpractice backlash against physicians and hospitals, who may be perceived in litigation as having been more interested in income and profits than in their patients.

Will managed care survive? Should it survive? In 1991, the American Managed Care Association defined managed care as ". . . a comprehensive approach to health care delivery that encompasses planning, education; and monitoring, coordinating and controlling quality, access and cost, while considering the interests of patients, providers and payers." So far, critics contend, managed care has delivered only on controlling access and cost. Many MCOs are still developing patient-subscriber education programs and criteria for measuring the quality of care offered by managed care providers.

Managed care *can* survive, provided that; 1) physicians do not become pawns of third party payers; 2) third party payers do not forget that managed care's main resource is the medical care; 3) physicians remember that they provide the care in managed care; and 4) managed care organizations do not become the symbols of greed.

New and lingering malpractice concerns

Old claims issues survive: Almost annually, new potential liability concerns emerges Neonatal injuries and silicone breast implants are two recent high-visibility issues that have plagued physicians, hospitals and insurers. However, such high profile cases represent a relatively small part of the overall malpractice picture. The bread-and-butter cases for most plaintiffs' attorneys are virtually the same they have feasted on for decades; failure to diagnose breast, lung and colon cancer, failure to diagnose heart disease, failure to diagnose and treat infection, negligent prescribing, refilling or monitoring medications, and failure to obtain an informed consent for invasive procedures. The collateral issues that provoke or exacerbate malpractice litigation also have remained the same for decades: inadequate medical records, weak doctor-patient communication; and unfounded peer criticism.

There are scant signs that these perennial problems are fading. In fact, there is evidence that they are worsening: the average cost of claims in each of these categories is increasing, and while the frequency of claims rises and falls in predictable cycles, overall, there are more malpractice claims per 100 doctors today than there were 15-20 years ago. Does this bleak outlook suggest that resolving the malpractice crisis is a hopeless task? Certainly not. The anesthesiologists have shown that analyzing the causes of malpractice claims in their specialty, and developing and implementing meaningful and reasonable practice guidelines that help avoid patient injuries, are effective solutions to the "malpractice problem." Most other specialties have not taken this route, and therefore have not had the dramatic decline in claims statistics and insurance premiums the anesthesiologists have experienced.

Arbitration: where are the facts? Many plysicians and their trade associations have tried unsuccessfully to get Congress to enact legislation to take malpractice claims out of the courts and adjudicate them in "alternative dispute resolution" (ADR) programs. Many who opposes a single payer health care system claiming that it would create new bureaucracies and invite government interference into matters best left to the states, appear unconcerned about government intervention and the new bureaucracies that ADRs would require. By some estimates, ADRs would be as costly, if not more so, than the current tort system.

Supporters of voluntary arbitration of malpractice cases claim that replacing tort litigation with voluntary, binding arbitration will eliminate economic pressures to settle, lower liability costs and decrease litigation stress. Is binding arbitration a solution whose time has come? Proponents have not made the case that it is. No insurer organization that arbitrates medical malpractice claims has disclosed complete data about long-term experience with binding arbitration. Anecdotal reports from entities that routinely arbitrate malpractice claims indicate that plaintiffs prevail in arbitration far more often than they do in litigation, and that discovery and defense costs in both forums are about the same. On February 24, 1992, *American Medical News* reported that a senior official at the Kaiser Foundation Health Plan, one of the nation's largest HMOs, said that Kaiser loses about half of its arbitration cases, compared to 20% at trial. The spokesman could not say if , overall, arbitration was less costly than litigation. Arbitration does provide a more private forum for adjudicating claims than the courts, but troubling questions remain. Without convincing evidence that replacing the tort system, which traditionally favors medical defendants, will reduce litigation costs and treat defendants equitably, insurers are not likely to embrace arbitration or other "alternate dispute resolution" plans.

Telemedicine, defined as practicing medicine via electronic means, may be another new liability risk for physicians. Radiologists now are able to receive and interpret radiographic images at remote locations, and send opinions to colleagues via computer, fax or phone. Similarly, other specialists can consult or co-treat with physicians in distant locales. Several projects exist in which a physician at one location can "examine" and "treat" patients at another. Using two-way audio- and video-hookups, and assisted by nurses at the remote location, physicians can "make rounds," take histories, "observe" patients, give advice, and do virtually everything they could do in a face-to-face encounter, except touch. For communities that are not served by a resident physician, telemedicine is a godsend. Telemedicine's possibilities are unlimited. So is the potential for abuse. Legislatures in several states are now drafting laws to regulate the practice of telemedicine. It is not an easy task, in part because the scope of telemedicine has not been completely defined, and in part because of the complexities of interstate licensing, credentialing and monitoring of physicians and other teleprofessionals. Lawmakers must decide how and in which jurisdiction liability claims that arise from interstate telemedicine malpractice will be adjudicated. Insurers worried about telemedicine liability and litigation costs are developing underwriting guidelines for this new risk.

Genetic discrimination: Success in medical research has created a frightening paradox. On one hand, researchers have developed genetic tests that can give early warning to persons at risk for some forms of cancer and hereditary conditions, such as Huntington's chorea. Early knowledge of their susceptibility allows individuals and families to plan their lives and to make informed choices about marriage and parenting. On the other hand, there is deep concern about the unintended negative consequences of such genetic testing. Some health insurers say that a disease-carrying gene is a "pre-existing condition" which their policies exclude. Other companies refuse life or health insurance to essentially healthy individuals who are known to have a disease-carrying gene, whether or not the person is sick or disabled — and may never be. This new paradox is being called "genetic discrimination." Debate about the legal, social and ethical aspect of genetic research will intensify, as some Americans see dark parallels between the well-intentioned efforts of modern genetic research and the racially-motivated genetic and medical experimentation of earlier generations in this country and abroad.

''Curing" malpractice before the millennium

If physicians and hospitals in the next century expect to vanquish the malpractice crisis that has dogged them for half of the current century, they will have to acknowledge and respond to the historical reasons people sue. Medicine does best when it is challenged. Health professionals must accept the challenge and approach malpractice as a disease. Its etiology and symptoms are well-known, and effective "treatments" are well-defined. It is essential that physicians find permanent relief from this debilitating problem. In the new millennium, their time, energy and expertise will be needed in debates about end-of-life care; genetic testing; substance abuse; domestic violence; and AIDS. These medical/social issues threaten the nation's health, and are likely issues in future malpractice litigation.

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(continued from page 104 - Evaluation and Treatment of Congestive Heart Failure)

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(continued from page 104)

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BOOK REVIEW

Frontier physician. The life and legacy of Dr. C. Earl Albrecht,

by Nancy Jordan. Fairbanks: Epicenter Press, 1996.
223 pages. Hard-cover Limited Edition $25.00;
Paperback $14.95

Probably no single physician in Alaskan history has had a greater impact on the health of its people—indeed the peoples of the North generally—than Dr. C. Earl Albrecht. Beginning as the first and only physician to the Matanuska Colony farmers in 1935, he went on to serve as the Alaska Command Surgeon for the U.S. Army during World War II. In 1945 Governor Ernest Gruening appointed him Alaska's first full-time commissioner of health, a post he held with distinction until 1956, leading the assault on the major health problems of the territory, particularly tuberculosis. For the next few years he held administrative and academic positions in Ohio and Pennsylvania, then returned to Alaska in 1971 in private practice. During his later years in Alaska, he conducted a significant and far-ranging survey of the impact of alcohol abuse in Bush Alaska.

Albrecht, more than any individual, was the founder and chief promoter of the international circumpolar health movement, having organized and led the first such conference of circumpolar nations at Fairbanks in 1967, and attending all subsequent conferences as his health allowed. In 1980 he founded the American Society for Circumpolar Health, which continues as the major force in organizing and supporting these conferences, the tenth of which was held in Anchorage in May of this year. In 1988 he was the first Alaskan to be awarded the Jack A. Hildes medal for his contributions to circumpolar health.

This new biography, by Nancy Jordan, is an informal narrative of Albrecht's life. It traces the story of the man, his times, and his accomplishments in generous detail. Albrecht's story is one that should be read by every Alaskan physician, since it is very much the story of Alaska's achievements in health over the past 60 years. The older generation, many of whom know the man personally, will appreciate the chance to reflect on an exciting period of history, while those of the younger set need to know, in this molecular biology generation, something of the giants on whose shoulders we stand as physicians in Alaska.

Dr. Albrecht himself, an honorary member of the Alaska State Medical Association, now lives in Bradenton, Florida, where at the age of 91, he is enjoying his well deserved retirement.

All proceeds from the hard-cover edition, incidentally, go to the Albrecht-Milan Foundation of the American Society for Circumpolar Health (P.O. Box 92419, Anchorage, AK 99509-2419), a nonprofit corporation which promotes health research and service among the peoples of northern countries. The book may be ordered from the foundation directly.

Robert Fortuine, MD

LETTER TO THE EDITOR

Problems with alcohol or depression?

Dear Editor:

If you feel that you may have a progressive, familial, metabolic disease, would you order tests on yourself, or get a real diagnostic evaluation from another doctor? What if this disease were fatal if left untreated? Would you procrastinate, or go Outside for the best consultation available and follow their treatment recommendations? The metabolic disease of alcoholism is such a killer.

In this issue is an advertisement for Abbott Northwestern Hospital in Minneapolis. ANH has no vested interest in making any diagnosis, as they have no treatment programs of their own. Their one week, voluntary, outpatient evaluation can determine if you do (or do not) have a problem with alcohol, depression, oppositional behavior, or other disorder that could benefit from treatment. Not all heavy drinkers are alcoholics - the alcoholics will go on to die while destroying their family.

The Physician's Assistance Committee knows of approximately 12 physicians who are living well with their recovery programs; but statistically there are approximately 90 more who need treatment. We wish that all alcoholic medical professionals could receive excellent treatment, but treatment first requires a diagnosis. Read the ad carefully for it may save your life.

Clinton B. Lillibridge, MD
Chairman,
Physician's Assistance Committee

GLIMPSES OF ALASKAN MEDICAL HISTORY

Edited by Robert Fortuine, MD

Traditional remedies of the Yukon-Kuskokwim Delta (1842-1844)

Lavrentii Alekseevich Zagoskin (1808-1890) was a Russian career naval officer who joined the Russian American Company in 1838. The first few years of his service in Alaska saw him commanding various ships ferrying supplies and personnel among the major Russian settlements. In 1840 he proposed an expedition to explore the interior of Alaska and in 1842 was sent by Governor Etholin to St. Michael, where he was ordered to conduct a survey into the largely unknown (to the Russians, at least) Yukon-Kuskokwim region. Over the next two years, he journeyed the middke and lower reaches of the Yukon, the lower Koyukuk, and the middle and lower Kuskokwim rivers. Throughout, he made detailed observations and collected ethnographic information on the Natives peoples he encountered, most of them meeting Europeans for the first time. He published his findings in Russian in 1847-1848 in a delightful and informative book which appeared for the first time in translation in 1967.

In the segment cited below, Zagoskin is writing of the coastal Eskimos. "Navigation Officer Vasilev," whom he quotes, was Ivan Iakovlevich Vasil'ev (1797-1838?), who in 1829-1830 explored the upper regions of the Nushagak River and its tributaries, then briefly passed over into the Kuskokwim River drainage.

Traditional healing among most Alaska Native groups was composed of two more or less distinct strands: shamanism, or magico-religious practice, which involved spirits, emotion, ceremony, and the supernatural; and empirical practice, which was based on the observed healing properties of plants, animals, and minerals. Although to a certain extent these traditions overlapped, the two types of practitioner were usually distinct. Shamans, however, sometimes used herbs and other empirical remedies, while healers also might ascribe supernatural powers to their practical remedies. In the passage below, Zagoskin describes a snippet of the empirical tradition; in the next issue of "Glimpses," he gives an example of shamanistic practice.

In the present segment, it is interesting to note that the Russians were willing to learn from the Native healers, even to the extent of submitting one of their paddlers to the knife. Another notable observation is that the Kuskokwim Natives suffered extensively from consumption, although contact with Russians to that time had been minimal, or at least spotty.

"The most prevalent diseases among the natives are abscesses, eye disease, and consumption. Since the Russians settled among the coastal peoples, the first stages of syphilis have appeared. It would be beneficial if completely healthy people were appointed to serve in distant provinces. Navigation Officer Vasilev notes that this scourge of humanity has also been communicated to the Aglegmyut. So far, venereal disease is unknown on the Yukon or Kuskokwim.

"The shamans treat all ailments with either spells or herbs. . . .

"The Russian traders have learned from the natives, and for chest pains and rheumatism they use with success an infusion of castoreum [beaver musk] in water or rum. I saw one of them, however, who made use of this carelessly, and who fell ill with jaundice. Also, they insist that a dried, ground beaver penis cures gonorrhea.

"Puncturing a place that is painful is as customary with the coastal people as with the Aleut and Kadyak natives. Vasilev writes that the Kuskokwim natives put fresh, raw liver on a frostbitten limb; on wounds they sprinkle burned hair—he does not say from what animal. Rashes and scabs they treat with otter dung. He also adds that during a trip on the Kuskokwim one of his paddlers suffered from such an extent from swollen testicles that they feared for his life. The shaman made the man well again for three leaves of tobacco; he punctured the swollen member and, while Vasilev was watching, drew from it some sticky fluid."

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From Out of the Past...

Gloria Park, MD

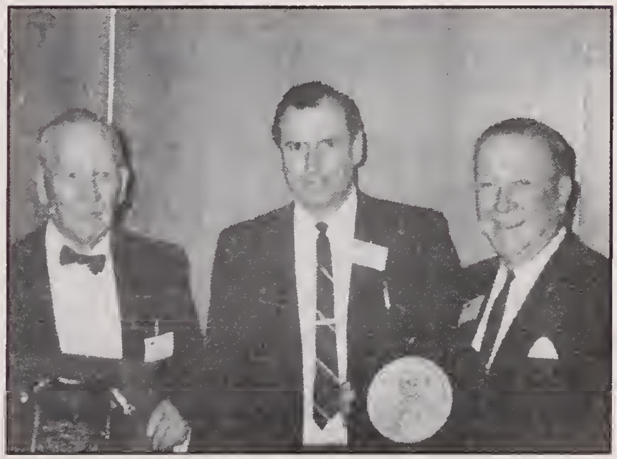
Anchorage Westward Hotel, scene of 21st Annual ASMA Convention 1966. 167 physicians registered.

Robert Shuler, MD, of Sitka — 1966 ASMA President

Robert Wilkins, MD, Pres-Elect. Also recognized for his 12 years as Secretary-Treasurer and Executive-Secretary for ASMA.

James Lundquist, MD of Fairbanks, Vice President

A. C. Chalmers, MD, Secetary-Treasurer



Dr Chenoweth, Morgan and Whitehead. Dr. Charles Chenoweth was namedPhysician of the Year. Dr. William Whitehead Community Service Award and 23 years as secretary to the State Medical Board.

SCIENTIFIC ARTICLES

Pheochromocytoma — Grace Jansen, MD, Warren Jones, MD, Theodore Shohl, MD

Reno-Vascular Hypertension — Michael Hein, MD

Scoliosis: Disease or Inheritance — William J. Mills, MD

Enteropathogenic E. Coli in Alaska — George Brenneman, MD and Robert Fortuine, MD

Otitis Media in Urban Alaska — Dwayne Reed, MD and Jacob Brody, MD

Empyema in Alaska Native Children — George Brown, MD

Use of Devil's Club in SE Alaska — James Justice, MD

Excerpts:

Devil's Club is the common name for Fatsia horrida, the family is Ginseng or Araliaceae. Other plants in the same family are English Ivy and Virginia Sarsaparilla. Stalks may be as big as 1 1/2 inches in diameter and reach eight feet in height. They are covered with sharp spines that can be very irritating upon contact. The extract has been used orally and in baths. It has been used as a perfume, emetic, lactation suppressant, for menstrual cramps, colds, arthritis, black eyes, gallstones, stomach ulcers, constipation and as a general tonic. The raw inner bark has been used on open wounds and in tooth cavities for pain.

EDITORIALS AND REPORTS

Good report on the status of air pollution in Anchorage by John Hickey. The charts cover 1953-1963. Population of Anchorage in 1960 was 44,625.

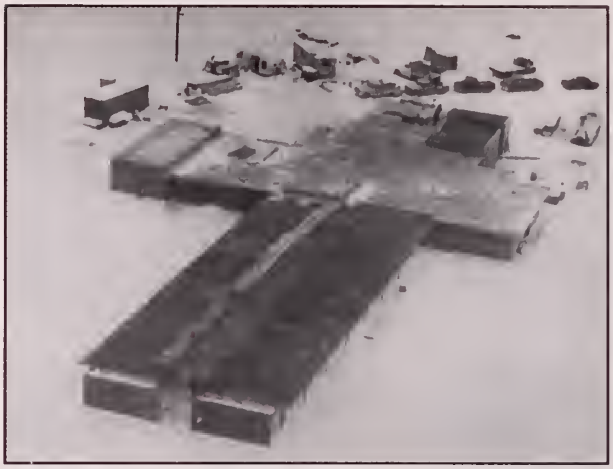
Appointed to the University of Alaska Board of Regents was Dr William Whitehead of Juneau and Dr Arthur Schaible of Fairbanks.

Dr Mike Beirne and Dr Milo Fritz have joined Dr Hoaglund as State Legislators.

Michael F. Beirne, MD

Dr. Arndt von Hippel reported on the television series put on by ASMA and the American Medical Society on Channel 11, KTVA.

The 30 bed Peninsula Hospital is becoming a reality.



The Arctic Health Research Center established in 1948 is in the process of moving from Anchorage to Fairbanks near the University of Alaska campus.

A grant of $213,998 has been announced by the National Institutes of Health to link the states of Washington and Alaska in an attack on deaths from heart disease, cancer and stroke.

Dr. Shuler announced the appointment of Mr. Robert Ogden as executive secretary for ASMA.

[*The following report is presented in toto because of the historical mileposts presented regarding the laws governing medical practice in Alaska and licensure.*]

Milo H. Fritz, MD

[The following report is presented in toto because of the historical mileposts presented regarding the laws governing medical practice in Alaska and licensure.]

A Review of Legislation Governing Medical Practice in Alaska

by
W.M. Whitehead, MD and John J. Dalton, MD
Juneau, Alaska

The first law governing medical practice in Alaska was quite simple, consisting only of a man's conscience and ublic opinion. Official regulation began in 1909 when Congress enacted legislation which provided that the graduate of any legally charted medical school which complied with the standards set forth by the Association of American Medical Colleges could be licensed by having his diploma recorded with the Clerk of the District Court of the judical division in which the applicant proposed to practice. The law further provided licensure for anyone who had been in practice for the three years prior to enactment regardless of the applicant's previous formal training in medicine. No provision was made for verification of an applicant's credentials and in severa instances they were found to be fraudulent after a license had been issued. During the four years, 1909-1913, about 150 physicians, many of whom were non-residents, were issued licenses to practice medicine and surgery in the Territory.

The First Territoria Legislature, meeting in 1913, established the Board of Medical Examiners. The members were appointed by the Governor for an indefinite term and did not require legislative confirmation; they also served without pay. The first Board appointed by Hon. J. F. A. Strong were as follows: W. G. Cassels, M.D., Fairbanks; Harry C. DeVighene, M. D., Juneau; Issac H. Moore, M.D., Ruby; J. H. Mustard, M.D., Nome; John L. Meyers, M.D., Ketchikan; Joseph H. Romig, M.D., Seward; J. M. Sloan, M.D., Nome; and David H. Sleem, M.D., Valdez.

At the first regular meeting of the Board held in Juneau July 8, 1913, Dr. I.H. Moore of Ruby was elected president and Dr. Harry DeVighne of Juneau, secretary-treasurer.* The death of Dr. D. H. Sleem of Valdez and emigration of Dr. W. G. Cassels of Fairbanks created two vacancies within the next six months. These were filled by Dr. Charles Winans, Valdez, and Dr. J. A. Sutherland, Fairbanks.

The function of the Board was to examine the credentials of a prospective licensee and, if properly accredited, issue him a temporary permit. A temporary permit was good until the next regular meeting of the Board (January or July) at which time the applicant had to present himself for examination in those subjects prescribed by law.

In creating the Board, the Legislature unfortunately failed to appropriate money for its operation. An initial fund of $145.00 was collected for the purchase of license blanks, stationery, etc, by collecting a fee of $5.00 from all physicians practicing in the Territory and eligible for licensure under the new law. Expenses of Board meetings were met from the candidates' examination fee of $25.00.

The second meeting of the Board was also held at Juneau in January of 1914. Fifty physicians who had been licensed by their respective Clerk of the Court or who had practiced in the Territory the required three years were granted licenses without examination.

Ballance, W. P.—Craig
Baines, M. D.—Wilmington, Del.
Baughman, J. A.—Seward
Benawa, W. G.—Douglas
Boyle, W. G.—Valdez
Brown, Bruce H.—Nulato
Campbell, E. O.—Craig
Carmichael, A. B.—Seattle
Craig, Harold M.—Haines
Danforth, Clarence E.—Chatanika
Dawes, L. P.—Wrangell
DeVighne, Harry C.—Juneau
Dickinson, Beatrice P.—Ketchikan
Dickinson, George L.—Ketchikan
Ellis, R. V.—Comet
Evans, Melville J.—Fairbanks
French, L. H.—Dillingham
Gable, W. G.—Skagway
Goddard, F. L.—Sitka
Green, W. F.—Tacotna
Hall, Mahlon F.—Fairbanks
Haxby, Thomas A.—LaTouche
Kyvig, K. A.—Knick
Lamb, Frank W.—Nulato
Midford, G. F.—Douglas
Miller, Thatcher—Ketchikan
Mustard, John H.—Nome
Myers, Ben. L.—Ketchikan
Meyers, John L.—Ketchikan

Palmer, A. J.—Porcupine
Payzant, James—Kennicott
Piperno, R.—Hector
Pryor, Lee—Petersburg
Robinson, E. B.—Nushagak
Romig, Joseph H.—Seward
Sargeant, Albert R.—Douglas
Schaleben, Henry O.—Seward
Schrock, E. B.—Kennicott
Silverman, Joseph A.—Kodiak
Simpson, James K.—Juneau
Skoonberg, A. W.—San Francisco
Sleem, David H.—Seward
Sloan, John M.—Nome
Smith, L. V.—LaTouche
Story; Henry G.—Ketchikan
Sutherland, J. A. —Valdez
Vance, Joseph A.—Cordova
Winans, Charles A—Valdez

These licenses were un-numbered as they were issued by the Clerk of the Court in various localities throughout the Territory. Official numbering began after this second meeting of the Board. Less than 100 licenses were issued over the next twenty years. As of this writing, the last issued license, No. 547, was obtained by Dr. Goerge H. Logenbaugh, Chief of Surgery, USPHS Hospital, Mt. Edgecumbe.

We thought it wuld be of interest to record the names to whom the first twenty licenses were issued. For reasons unknown there were two number 2's, and numbers 11, 17, and 18 are missing.

During the years that followed,, the law was revised and amended as needed to clarify conflicting or ambiguous passages and more effectively meet the changing needs and desires of modern day medicine.

1917 a) An internship for one year in an accredited hospital or four years of active practice were added to the requirements for licensure.
b) Standards for licensure by reciprocity were instituted and the fee set at $25.00.
1921 a) The fee for reciprocal licensure was raised to $100.00
b) The Legislature appropriated $500.00 per biennium to fund the Board expenses. All monies collected were paid directly to the Territorial Treasurer.
1929 a) Membership representation was defined by law which allocated two members from the First Judicial District and one each from the other three Judicial Districts.
b) Times for examination were changed from January and July to the first Tuesday in March and September. the Board has since changed its policy to the present system, i.e., examination at any time.
1931 An occupational tax of $10.00 per annum was levied against all licentiates regardless of residence.
1946 Basic Science Certification was added to the requirements for licensure and a Board of Examiners in the Basic Science created. The five members appointed by the Governor serve four year terms without compensation. Unlike the Board of Medical Examiners, these appointments require legislative confirmation. Law decrees that the five members consist of a layman (and automatic chairman), two doctors of medicine, one doctor of chiropractic, and one doctor of osteopathy. The laymember is automatically chairman while the secretary-treasurere is electede by the members of the Board. Original members appointed by Governor Ernest Gruening were 1) Don Skuse, Juneau,

Number	Place	Issued	Name	Residence in 1932
1	Juneau	1914	W. A. Myers	Kansas City, Mo.
2	Juneau	1914	H. E. Auringer	Addison, N.Y.
2	Juneau	1914	Wm. Pallisler	New York
3	Juneau	1914	E. O. Campbell	Tonasket, Wash.
4	Juneau	1914	E. M. Bevis	Goleta, Calif.
5	Juneau	1914	Lorenzo Smith	LaTouche, Alaska
6	Seward	1914	James G. Anderson	Petaluma, Calif.
7	Seward	1915	Ezra Brooks	Berkeley, Calif.
8	Fairbanks	1915	E. S. Reedy	Seattle, Wash.
9	Seward	1915	Herbert Stoughton	Malone, N.Y.
10	Juneau	1915	M. F. Spier	Unga, Alaska
11				
12	Juneau	1916	David H. More	Long Beach, Calif.
13	Juneau	1916	Lester C. Smith	Grass Creek, Wyo.
14	Fairbanks	1916	Lillian G. Stevenson	unknown
15	Juneau	1916	J. L. Bulkley, J.	Richmond Hill, N.Y.
16	Juneau	1916	Douglas Brown	Rutland Heights, Mass
17				
18				
19	Juneau	1916	Charles C. Benedict	Whipple, Arizona
20	Seward	1916	John Bradley Besson	Wooster, Ohio

Chairman; 2) Earl Albrecht, M. D., Juneau, Secretary-Treasurer; 3) A S. Walkowski, M. D., Anchorage; 4) Howard Vance, D. O. Juneau; and 5) George Caldwell, D. C., Juneau. The present Board, appointed by Governor Egan, consists of Ralph Wright, Juneau, Chairman; John J. Dalton, M. D., Juneau, Secretary-Treasurer; A. S. Walkowski, M. D., Anchorage; Richard Sutherland, D. O., Anchorage; and C. O., Risch, D. C., Anchorage.

1957 Physical Therapists were placed under jurisdiction of the Board of Medical Examiners.

1966 License to practice medicine and surgery by Doctors of Osteopathy was authorized by the 1966 Legislature. Practice in the state for one year prior to January 1, 1966, or successful completion of examinations in the Basic Sciences and before the Board of Medical Examiners are the requirements. Currently, two licenses have been authorized, both under the "grandfather clause."

The present Board of Medical Examiners appointed by Governor Egan,. is composed of H. I. Akiyama, M. D., Juneau, President; W. M. Whitehead, M. D., Juneau, Secretary-Treasurer; Charles F. St. John, M. D., Anchorage; and Raymond D. Evans, M D., Fairbanks.

Acknowledgements:

Tremendous assistance was given by Mrs. Dorothy Russell and Miss Marian Davis.

Muktuk Morsels by Arndt von Hippel, MD

Dr Helen Whaley is taking a one-year fellowship in Pediatric Neurology at Stanford.

Dr William James has returned to Fairbanks after completing a pediatric residency.

Dr Edward Meyer has left Fairbanks and the following have arrived in Fairbanks - Drs Joseph Worrall, Lawrence Dunlap, John Noyes and Martha Kowalski.

Dr. Herb James has moved from Seward to Anchorage. Dr. Ed Voke has returned to Anchorage after an orthopedic residency and Dr Paul Dittrich is also a new orthopedist in Anchorage.

Dr William Mills has enlisted for two years in the Navy and is chief of orthopedics in DaNang, Viet Nam. He then plans to spend six months in the Antarctic.

Babies have arrived in the homes of Drs Shirley and Robert Fraser, Dr Hein and Dr Marcell Jackson.

The Mayo Cardiac Clinic team visited Sitka, Anchorage and Fairbanks.

In Memoriam — William M. Whitehead, MD

Died of a cerebral hemorrhage November 12, 1966 while at his hunting cabin on Chichagof Island. He interned at Virginia Mason Hospital and arrived in Wrangell in 1934. He married the school teacher there and then moved to Juneau. He was a graduate of the University of Virginia School of Medicine in 1931. He was very active in both community affairs and the medical profession.

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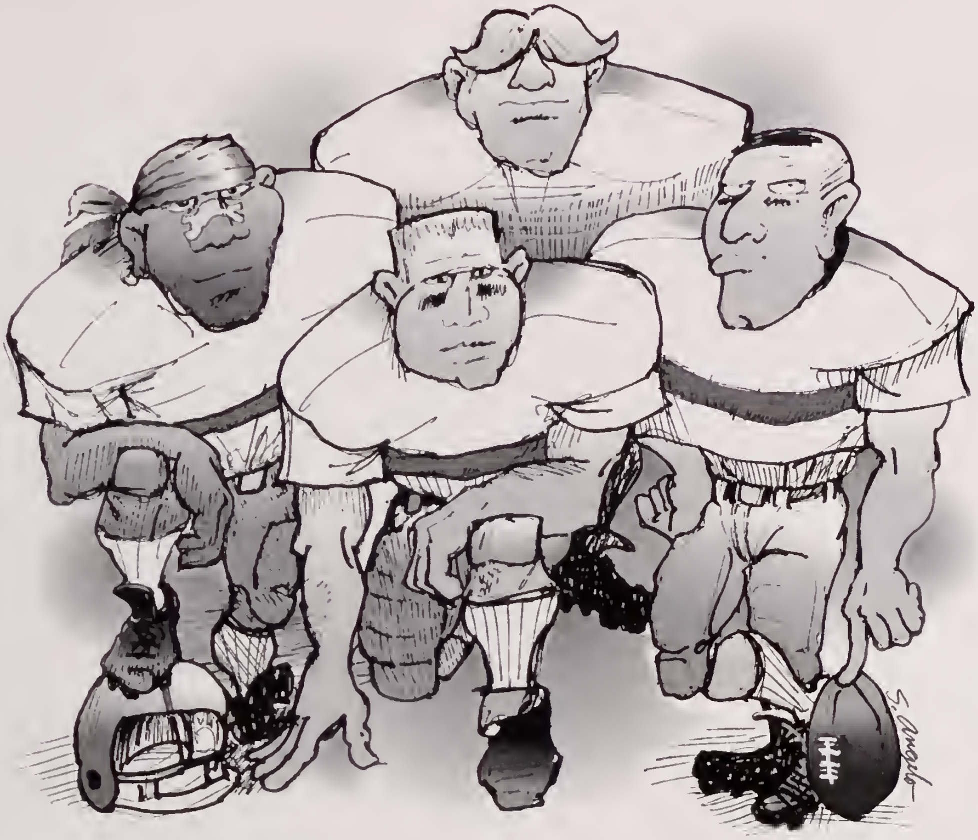

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About the cover: Tanaga Island — Aleutian Islands

Tanaga has three volcanoes. The easternmost was on the cover of the last issue - these are the two westernmost. The one on the left is the tallest - 5925 feet MSL.

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WHAT DETERMINES SURVIVAL AND MORBIDITY AT VERY EARLY GESTATION ? ALASKA'S EXPERIENCE

Jack Jacob, MD[1]

ABSTRACT

The charts of all infants (N=46) 22-25 weeks gestation admitted to the Newborn Intensive Care Unit at Providence Alaska Medical Center from 1991-1994 were reviewed to determine factors associated with mortality and severe morbidity. Survival rates at 22 & 23 weeks gestation was poor (25% and 14% respectively). Survival rates of 24 and 25 week gestation infants were similar (53% and 62% respectively). Infants with "poor" outcomes (death or survival with Grade 3 or Grade 4 intraventricular hemorrhage or periventricular leukomalacia) were more likely to have suboptimal intrapartum care which primarily involved delayed intervention on behalf of the fetus. This was manifested as perinatal asphyxia needing vigorous resuscitation at birth with cardiac compression and epinephrine use, and the development of persistent pulmonary hypertension of the newborn in the first 24 hours of life. Ten of the fifteen deaths were primarily due to intractable respiratory failure complicated by barotrauma from ventilator therapy.

The fetus at 24 and 25 weeks should be considered viable. Improving the survival and morbidity of these extremely low birthweight babies will require us to optimize fetal care in complicated pregnancies at previable and very early viable gestations (20-25 weeks).

INTRODUCTION

There has been a major improvement in neonatal mortality in the last decade. The most current information shows that Alaska's neonatal mortality for 1993 is 4.3 per thousand live births, down from 6.3 in 1985 (1). This trend has been associated with improved birthweight specific mortality, especially for the very low birthweight preterm infant. This period has also seen advances in our understanding of the physiology of the fetus and tiny preterm infant, and the introduction of therapeutic advances such as the intensive management of high-risk pregnancies, antenatal corticosteroid use for accelerating lung maturity, surfactant replacement therapy for respiratory distress syndrome, and high frequency ventilation.

A recent American College of Obstetricians and Gynecologists Technical Bulletin suggested a current viability threshold of approximately 24 weeks gestation based on current information (2). It also recommended that these guidelines should be considered in light of the perinatal statistics of a region's own tertiary center. This review was therefore undertaken to look at the issue of survival and serious morbidity in the Alaska population of newborns of very early gestation (22-25 weeks gestation) with an emphasis on factors that may impact their survival and serious morbidity.

METHODS

Maternal and infant charts of patients admitted to the newborn intensive care unit at Providence Alaska Medical Center from 1991-1994 with gestational age by best obstetrical estimate of 22-25 weeks were reviewed. Patients meeting these criteria were identified using a database maintained by Alaska Neonatology Associates. Clinical information was abstracted to identify outcome variables and potential dependent variables. For purposes of the study a "good" outcome was defined as an infant who survived hospitalization and had no more than a grade 2 intraventricular hemorrhage (IVH) and no periventricular leukomalacia (PVL). A "poor" outcome was defined as an infant who either died, or survived with a grade 3 or 4 IVH or PVL, or needed to be discharged on a ventilator. Additional long term morbidity recorded was chronic lung disease requiring oxygen at the time of discharge. Lack of prenatal care was noted when prenatal care was sought just prior to delivery.

Differences between group outcomes were analyzed using analysis of variance.

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(1) Alaska Neonatology Associates and Providence Alaska Medical Center, 3340 Providence Dr. Suite 366, Anchorage, AK 99508.

RESULTS

The survival and major morbidity at various gestational ages is shown in Table I. Survival rates did not exceed 50% until 24 weeks gestation. Survival for infants at 24 and 25 weeks gestation were similar. Table II shows the comparison of infants with "good" and "poor" outcomes in terms of patient demographics and prenatal care. None of the differences are significant. Table III shows a similar comparison for intrapartum and delivery room care. "Good" and "poor" outcome groups were significantly different (P=.024). This was manifested as a higher incidence of suboptimal care in the "poor" outcome group. The single case of suboptimal intrapartum care in the "good" outcome group consisted of a precipitous delivery at the tertiary center without experienced resuscitation personnel present at the time of delivery. At Providence Alaska Medical Center a neonatologist is available in house and is usually present within a few minutes of such a delivery. In the "poor" outcome category suboptimal care consisted of the following: delay in performing a prompt cesarean delivery for fetal distress or fetal malposition (4 cases), precipitous delivery at the tertiary center (2 cases), mother kept in referring hospital with fever and amnionitis for a prolonged time prior to transfer (1 case), traumatic cesarean delivery associated with a severed umbilical cord (1 case), and underestimation of maternal dates resulting in suboptimal intervention on behalf of the fetus (1 case). Ironically, suboptimal intrapartum care did not result in a lower cesarean delivery rate in the "poor" outcome group. In fact the cesarean delivery rate in this group was surprisingly high at 67%. In several cases cesarean delivery was delayed until transfer to the tertiary center was accomplished. This implies that the cesarean delivery was done too late for optimal fetal benefit. Consistent with this supposition was the need for cardiac compression and epinephrine use for resuscitation in 24% of the "poor" outcome group. Problems in delivery room care consisted of delays in immediate tracheal intubation due either to the absence of appropriate personnel at the delivery or technical difficulty in tracheal intubation. None of the other differences between groups were significant.

Table IV shows differences between groups in relationship to issues in medical care. "Good" and "poor" outcome groups were significantly different (P=.019). Unexpectedly, we found persistent pulmonary hypertension of the newborn (PPHN) on the first echocardiogram done at <24 hours of age in 24% of infants with "poor" outcomes; PPHN was absent in the "good" outcome group.

There were a total of 15 deaths. Of these 10 were primarily respiratory in nature consisting of severe respiratory failure unresponsive to conventional therapy. Complicating factors included the development of airleaks from ventilator-induced barotrauma used in an attempt to manage the respiratory failure. There were 2 cases of gastrointestinal deaths (necrotizing enterocolitis, bowel perforation), one case of severe unresponsive metabolic acidosis associated with perinatal asphyxia, and one case of superior vena cava syndrome resulting from a central venous catheter thrombus.

DISCUSSION

Traditionally, we have defined the limits of viability at Providence Alaska Medical Center at 25 weeks gestation while recognizing that over the last several years we have had an increasing number of survivors at 24 weeks gestation. Before recommending a formal change in practice it is important to look at results from our tertiary care center.

This review documents a similar survival in 24 and 25 week gestation infants that exceeds the threshold of 50% survival that is used to recommend vigorous intervention on behalf of the fetus (3).

TABLE 1.

SURVIVAL & MORBIDITY OF ELBW BABIES
1991-1994(N=46)

Gest. Age(N)	Survival(N)	Home oxygen*(N)	All IVH**(N)	Gr 3 & 4 IVH**(N)
22 weeks(N=4)	25%(1)	0%(0)	25%(1)	25%(1)
23 weeks(N=7)	14%(1)	100%(1)	14%(1)	14%(1)
24 weeks(N=19)	53%(10)	20%(2)	42%(8)	21%(4)
25 weeks(N=16)	62%(10)	30%(3)	75%(12)	44%(7)

*percent of survivors
**percent of survivors and non-survivors

TABLE II.

COMPARISON OF "GOOD" AND "POOR" OUTCOMES*,
24 & 25 WEEKS GESTATION:
DEMOGRAPHICS AND PRENATAL CARE

Parameter	"Good" outcome (N=14)	"Poor"outcome (N=21)
birthweight	713 gm (604-942)*	704 gm (380-1023)*
Alaska native	14% (2)	14% (3)
single birth	64% (9)	76% (16)
inborn	71% (10)	90% (19)
lack of prenatal care	21% (3)	14% (3)
maternal amnionitis	43% (6)	24% (5)
antenatal steroids	28% (4)	24% (5)

* mean (range)

TABLE III.

COMPARISON OF "GOOD" AND "POOR" OUTCOMES,
24 & 25 WEEKS GESTATION:
INTRAPARTUM AND DELIVERY ROOM CARE

Parameter	"Good" outcome (N=14)	"Poor" outcome (N=21)
suboptimal intrapartum care	7% (1)	43% (9)
cesarean delivery (C/S)	50% (7)	67% (14)
C/S requested by parent	21% (3)	14% (3)
neonatology at delivery	79% (11)	86% (18)
Apgar 1 min < 3	50% (7)	71% (15)
Apgar 5 min < 7	50% (7)	71 % (15)
CPR, epinephrine for resuscitation	0% (0)	24% (5)
delivery room care problems	14% (2)	10% (2)

TABLE IV.

COMPARISON OF "GOOD" AND "POOR" OUTCOMES,
24 & 25 WEEKS GESTATION:
ISSUES IN MEDICAL CARE

Parameter	"Good" outcome (N=14)	"Poor" outcome (N=21)
Primary lung disease		
RDS	43% (6)	71% (15)
accel. lung maturity	43% (6)	19% (4)
infection / RDS	14% (2)	10% (2)
Patent ductus arteriosus		
treatment < 24 hr of age	54% (7)	38% (8)
no treatment	15% (2)	10% (2)
Persistent pulmonary hypertension	0% (0)	24% (5)
Intraventricular hemorrhage		
grades 1-2	23% (3)	19% (4)
grades 3-4	0% (0)	52% (11)
Ventilator at discharge	0% (0)	0% (0)

In addition to survival, quality of life issues are important determinants in the decision for intervention on behalf of the fetus. With this in mind, "good" and "poor" outcomes in our population were chosen based on morbidity that frequently results in significant long term medical and developmental disability (4). The clinical items chosen for review were suspected to effect outcome of preterm newborns based on the medical literature. Generally, poor prenatal care, outborn deliveries, multiple gestation pregnancies, and an Alaska Native racial background are associated with higher neonatal mortality (5-8). The use of antenatal corticosteroid therapy has been associated with a lower mortality and a reduction in major morbidity such as RDS, intraventricular hemorrhage, and necrotizing enterocolitis (9). Experience suggests that maternal amnionitis is frequently associated with preterm labor at very early gestation (10), and that its presence is commonly associated with the absence of RDS and the presence of accelerated lung maturity. The absence of RDS frequently results in milder lung disease with a resultant improvement in survival. Finally, the early use of indomethacin for patent ductus arteriosus (PDA) closure has also been associated with an improved outcome in the very low birthweight infant (11).

When preterm delivery is imminent, decisions made about intrapartum care of the tiny fetus may also affect outcome (2). The most important finding in this review is that within this very early gestational age group the quality of intrapartum care had a profound effect on mortality and severe morbidity. This was reflected in a delay in prompt maternal intervention and postponement of surgical delivery of the fetus until transfer to the tertiary center occurred. Ironically, such an approach did not result in a lower cesarean delivery rate. This delay in prompt intervention on behalf of the fetus resulted in an infant with lower apgar scores, and a need for vigorous resuscitation with cardiac compression and epinephrine use. Furthermore, it is also likely that the accompanying perinatal asphyxia resulted in a high incidence of PPHN, a finding previously unreported under these circumstances in this gestational age group. This finding underscores the notion that all hospitals that offer delivery services need the capability to perform safe cesarean deliveries. This standard of perinatal care, widely accepted elsewhere in the United States, has never been embraced as a standard of care for all rural Alaskan hospitals. It also emphasizes that, although maternal transport is a desired standard of care in such situations its value should be weighed against the risks; transport should not interfere with the prompt initiation of therapy (2).

It is also apparent from this review that those of us involved in perinatal care at the tertiary center need to develop strategies to deal with the occurrence of precipitous deliveries in order to ensure immediate skilled resuscitation in these fragile patients.

It is likely that the suboptimal care identified in this review is a culmination of suboptimal care provided for women at early gestation who develop complications of pregnancy. Such a phenomenon is not unusual during periods of advancement in perinatal care. This is currently relevant since we have recently entered an era of advanced perinatal care with the establishment of feto-maternal medicine as a specialty in Alaska.

A recent clinical case aptly describes the differences in outcome and quality of life that are achievable: A 24 year old gravida 2 para 1 white female presented to her primary physician in a rural Alaskan community at 22 weeks gestation with cervical dilatation. Initially she was kept in her community and was not felt to have a viable fetus. The following day, after consultation with feto-maternal medicine, she was transferred to Providence Alaska Medical Center. She had a circlage placed immediately and was managed aggressively in the hospital with tocolysis. She was Group B Streptococcus negative but Ureaplasma positive on two separate occasions. On each occasion she was treated with erythromycin. She maintained her pregnancy in the hospital until 26 weeks gestation when she ruptured her membranes spontaneously. The circlage was removed and she delivered shortly thereafter an infant weighing 950 grams. Although the infant did not have RDS, she needed high frequency ventilation followed by conventional mechanical ventilation for 10 days prior to extubation. The respiratory course was complicated by a PDA treated with indomethacin on the first day of life. Because of maternal history the infant was treated with erythromycin for one week. Infant Ureaplasma cultures were negative. Tracheal cultures at birth grew yeast associated with pneumonia, and the infant was treated with amphotericin. Other problems encountered during the hospital stay were apnea of prematurity and nosocomial coagulase negative staphylococcus pneumonia. No intraventricular hemorrhage or periventricular leukomalacia were observed. The infant was discharged at day 82 of life without any respiratory problems and a normal neurobehavioral examination.

This case, although anecdotal exemplifies the impact that current feto-maternal care can have on mortality and the quality of life of extremely low birthweight survivors.

It is important to note that none of the information presented here changes the important role that parents have in the combined decision making with their primary physician, perinatologist, or neonatologist. In the process of doing this it is important to have current and factual information for parents to make their decision. The underestimation of neonatal survival at multiple gestational ages has been well documented (12), and may result in overly pessimistic information being given to parents. This is a difficult issue to deal with because obstetric perceptions about viability may become self-fulfilling due to a lack of willingness to intervene on behalf of the fetus. In fact, when birthweight and gestational age are controlled, fetuses considered viable are 18 times more likely to survive than those considered non-viable (13). In my experience it is unusual for a physician's discussion with parents about viability to include information on the effect of their decision making on behalf of the fetus on outcome.

An important medical issue uncovered in this review is the high incidence of PPHN in the group of infants with perinatal asphyxia. PPHN has been described in term or near term infants (14), although this view has been challenged (15). PPHN is a difficult disease to treat, frequently requiring high ventilator pressures to overcome hypoxia. The extremely low birthweight (ELBW) baby is particularly vulnerable to fail such traditional management because of their propensity to pulmonary barotrauma. The high number of respiratory deaths and frequent finding of pulmonary interstitial emphysema and airleaks in these infants suggest that PPHN is an important determinant in the poor outcome of these infants.

There are two approaches in dealing with this problem. First, it is likely that a more vigorous approach in preventing perinatal asphyxia is likely to prevent this problem. Secondly, newer approaches in the treatment of PPHN such as inhaled nitric oxide therapy may be of use (16). Although the FDA has not approved nitric oxide for general use, Alaska Neonatology Associates and Providence Alaska Medical Center currently has FDA approval for its investigational use that includes this category of neonate.

In summary, it is my recommendation that the threshold for viability be moved to 24 weeks gestation. In addition, we should optimize fetal care in complicated pregnancies at previable and borderline gestations (20-25 weeks). This includes early consultation with feto-maternal medicine so that fetal care can be optimized at the tertiary center. I believe that the payoff for this approach will not only be improved survival, but also a better quality of life for survivors.

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A Clinical Intervention For Children Exposed to Alcohol In Utero

Carl Li, MD, MPH(1)
Karen Pearson, MS(2)

ABSTRACT

This study describes a statewide system of delivering special services to children affected by prenatal alcohol exposure and investigates the usefulness of this system as part of a statewide fetal alcohol syndrome surveillance system.

The Alaska Department of Health and Social Services administered seven clinics in four of the five largest cities in Alaska during 1992-1995.

Among the 74 children selected to be evaluated for fetal alcohol syndrome, 70 had one or more central nervous system abnormalities. Four children were assigned the diagnosis of fetal alcohol syndrome. Recommendations made to parents of the evaluated children included parent support group follow-up (38%), referral to a psychologist (35%), and further school testing and planning (27%).

Clinics for alcohol-exposed children provide health officials an opportunity to deliver appropriate services to children exposed to alcohol in utero and to their families. Further studies are necessary to improve the delivery of services to birth mothers of alcohol-exposed children who attend the clinic. Statewide fetal alcohol syndrome surveillance can be accomplished by linking these clinics with other state data sources.

INTRODUCTION

Fetal alcohol syndrome (FAS), characterized by growth deficiency, central nervous system abnormalities, and a characteristic set of dysmorphic facial features (1-4), is considered one of the most common preventable causes of mental retardation in the United States (5). Estimates of the annual costs associated with FAS in the United States range from $74.6 million (6) to $321 million (5), depending upon the population estimate of the incidence of FAS. The prevention of FAS has become a national priority (7). Among the 50 states, Alaska has one of the highest prevalence rates of binge drinking or drinking more than 30 drinks per month among women of reproductive age (8,9).

In 1991, the Alaska Department of Health and Social Services, with the support of the Centers for Disease Control and Prevention through an interagency agreement, decided to develop a statewide system for the delivery of special services to children affected by prenatal alcohol exposure and to develop a component of a statewide surveillance system for FAS (10). The Alaska Department of Health and Social Services developed a population-based approach to link the delivery of special health services to children affected by in utero alcohol exposure with statewide FAS surveillance activities. This approach involved a series of newly established FAS diagnostic clinics that the Alaska Department of Health and Social Services conducted in various communities across the state. The FAS clinics were designed to identify children with FAS through active case-finding, to refer them to appropriate services, and to coordinate the care of all children evaluated at the clinic. This report describes the operation of a statewide system of delivering special services to children affected by prenatal alcohol exposure and investigates the usefulness of this system as part of a statewide FAS surveillance system. Because many birth mothers of children exposed to alcohol in utero attended the clinics and the concern that these mothers might continue to drink, the characteristics of those birth mothers of alcohol-exposed children are described.

METHODS

Seven State of Alaska diagnostic clinics were conducted in four of the five largest cities in Alaska (11) during 1992-1995. These four cities accounted for 69% of persons 0-19 years old in the state (11). Each diagnostic clinic was conducted over a two-day period (except for one clinic in 1994 which was conducted in one day). The clinic visit consisted of two parts. During the first part, the child was evaluated for FAS by either a board-certified dysmorphologist or a developmental pediatrician. During the second part, the medical, psychological, and educational needs of the child and parents were assessed, and a service referral plan was developed. The clinics were conducted in accordance with Alaska Department of Health and Social Services confidentiality guidelines.

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(1) Division of Birth Defects and Developmental Disabilities, National Center for Environmental Health, Centers for Disease Control and Prevention, 4770 Buford Hwy NE, Mailstop F-15, Atlanta, GA 30341.

(2) Division of Public Health, Alaska Department of Health and Social Services, Anchorage, Alaska.

Subject Selection

Because the Alaska Department of Health and Social Services offers many services focusing on children 0-5 years old, the clinics initially targeted children in this age group. The upper age limit was increased to 19 years after the second clinic because clinic staff found that school-aged children were being regularly referred to the clinic for evaluation of emerging problems.

Children were referred to the clinic through a two-stage process. In the first stage, state and local community-based agencies and programs were asked to screen their case files for children who had documented evidence of exposure to any amount of alcohol in utero and who had either growth deficiency or central nervous system impairment. Exposure to alcohol in utero was included as one of the screening criteria to ensure that FAS could be a possible diagnosis. Growth deficiency and central nervous system impairment were not evaluated in detail at this stage.

The state and local agencies and programs which were contacted included the following: the state's early intervention program for children 0 to 3 years of age (described in Part H of public law 99-457, Individuals with Disabilities Education Act), child protective services program, public health nursing program, Head Start, and the Women, Infants, and Children program; regional Native health corporations and an Indian Health Service unit located in one of the four cities; a support group for parents of children diagnosed with FAS; and local school districts, adoptive agencies, and mental health resources. Pediatricians and family practitioners were contacted and encouraged to refer children who met the above mentioned screening criteria. In addition, newspaper and radio announcements promoted the clinics and encouraged parents to refer children who might meet the screening criteria. For each potential FAS child, the referral source completed a standardized referral form detailing the child's health information.

During the second stage, clinic staff used information on the referral form to select the specific children for clinic evaluation. Priority was given to children who met the following criteria: 1) evidence of prenatal growth deficiency (defined as birth weight adjusted for gestational age or birth length $\leq$10 percentile) or postnatal growth deficiency (defined as weight or height $\leq$10 percent adjusted for age and gender) and 2) an identified central nervous system (CNS) abnormality (including attention deficit disorder, hyperactivity, developmental delay, learning disability, behavioral deficits, and structural and neurologic abnormalities) (4). If the clinic still had space to accept more children for evaluation, priority was then given to children with evidence of either growth deficiency or CNS impairment. Because the referral sources had no training in the quantitative measurements of facial dysmorphology, the referral criteria excluded the cluster of facial features suggestive of FAS (which are defined as short palpebral fissures, an elongated midface, a long and flattened philtrum, a thin upper lip, and a flattened midface) (4). Children not selected for clinical evaluation were referred to other resources for further follow-up.

Clinic Evaluation

Because FAS can affect so many domains of a child's life, the clinics used an interdisciplinary approach in evaluating children and referring them to services. Before a child's evaluation, clinic staff members reviewed the child's medical records obtained from each child's physician for information on the child's past medical history, including data on birth, growth, development, and behavior. Clinic staff members also reviewed the child's school record obtained from each child's school teacher. In addition, the caretaker for each child completed a detailed history form, which included information about the child's health, development, behavior, and education; the birth mother's pregnancy and prenatal substance use history, if available; and the child's family history, if available.

During the clinic, each child received a two-part evaluation. First, a board-certified dysmorphologist or a developmental pediatrician obtained a brief history, performed a physical examination (which included measuring the child's current height and weight and conducting a detailed examination of the child's face), and assigned a final diagnosis to the child. During the second part, the child's health, psychological, behavioral, and educational needs were assessed by an interdisciplinary team in order to develop a comprehensive care plan.

The interdisciplinary assessment team consisted of the diagnosticians, a public health nurse, the child's early intervention program staff member or school teacher, and, if already working with the family, a child protective services case worker. Because local professional staff were not available at all clinic sites, a substance abuse counselor was included on the team at two sites, and a psychologist, a behavior and education management specialist, and a nutritionist were included at three sites. The children's private physicians were invited to be a part of the interdisciplinary team, but none of them participated in any of the clinics. The assessment team developed a care plan and assembled a community resource referral list that attempted to meet all of the identified needs of the parents and children. For parents, the resource list included parent resource centers, a support group for parents with FAS children, financial resources, and alcohol treatment programs. For children, the list included psychology resources, nutrition counseling programs, and local educational resources. The care plan and resource list were discussed with the family. A care coordinator for the family was selected from among the team members. The responsibilities of the care coordinator included coordinating the child's care plan, providing limited follow-up usually consisting of a telephone call, and linking the child and parents to other services.

Statistical Methods

The frequencies of demographic and clinical characteristics are presented for all children evaluated. The frequencies for maternal characteristics are presented for birth mothers living with their children at the time of the clinic visit. Frequencies were calculated using Epi Info Version 6.0 (12).

RESULTS

At the seven clinics, 182 children exposed to alcohol in utero and suspected of having FAS were referred for evaluation. In accordance with the priority criteria, 74 (41%) of the 182 referred children were selected for evaluation by the dysmorphologist or developmental pediatrician for FAS. The remaining 108 children were referred to other resources for further follow-up.

The ages of the 74 children evaluated ranged from two months to 16 years (median, 4.6 years); 65% were male; and 64% were Alaska Native, 31% were Caucasian, and 5% were African American. Children were most commonly referred by their parents or legal guardians (34%) (Table 1). Of the 74 children evaluated, only 26 (35%) were living with one or both of their birth parents.

Table 1. Patient Characteristics

Referral Source	**N**	**%**
Parents or legal guardians	25	34
Child protective services	16	22
Early intervention program	12	16
Private physicians	6	8
Public health nursing	5	7
School nurses	3	4
Alaska Native health corporation	2	3
Head Start	2	3
Indian Health Service	1	1
Local mental health agency	1	1
Women, Infants, and Children's program	1	1
Total	74	
Custody Status		
Birth parents	26	35
Children live with:		
Birth mother	16	22
Birth father	6	8
Both birth parents	4	5
Foster parents	25	34
Adoptive parents	20	27
Court-appointed legal guardians	3	4
Total	74	

Children's Characteristics

Prenatal or postnatal growth deficiency was noted among 42 (57%) of the children (Table 2). Their median gestational age at birth was 39 weeks (range: 30-42 weeks). Among the 74 children, 28% were preterm (<38 weeks). The median birth weight of the children evaluated was 2891 grams (range: 926-4309 grams). Fifteen percent of the children had a birth weight-for-gestational age $\leq$10 percent.

At least one facial feature of FAS was noted among 34 (46%) of the 74 children (Table 3). The physicians noted a cluster of facial features suggestive of FAS in 12 (16%) children.

Overall, 95% of the children had one or more central nervous system (CNS) abnormalities (Table 3). The CNS abnormalities most frequently noted in the medical and educational charts among the 74 children were developmental delay (74%) (which included 46% with fine motor delay and 34% with gross motor delay), short attention span (69%) (which included 20% with attention deficit disorder), speech or language delay (65%), hyperactivity (51%), and learning disability (36%). Additional CNS abnormalities included seizure disorder (8%) and hypotonia (7%). Thirty-one (42%) children had evidence of short attention span, developmental delay, and speech or language delay.

Among the 74 children evaluated, 54% had a history of neglect or abuse, including 28% and 19% with a documented history of physical or sexual abuse, respectively (Table 3).

Only four (5%) of the 74 evaluated children were assigned the diagnosis of FAS (Table 3). One child had been previously diagnosed by a pediatrician as having FAS. Three of these four children met either the height-for-age or weight-for-age criteria for FAS at the time of the clinic visit. The fourth child had a height-for-age of 38 percent and a weight-for-age in the 50th percentile at the time of the clinic visit. The diagnostician stated that this fourth child was "growth deficient" based on the

Table 2.

Physical Characteristics of 74 Clinic Patients

Postnatal Growth Deficiency[*]	N	%
Yes	39	53
No	35	47
Prenatal Growth Deficiency[+]		
Yes	18	24
No	44	59
Missing data	12	16
Prenatal or Postnatal Growth Deficiency		
Yes	42	57
No	32	43
Gestational Age (weeks)		
preterm (<38)	21	28
term (38-42)	33	45
missing	20	27
Birth Weight (grams)		
≤ 2680 (≤10 percentile)	24	32
> 2680	38	51
missing	12	16
Birth Length (cm)		
≤ 47 (≤10 percentile)	17	23
>47	26	35
missing	31	42
Birth Head Circumference (cm)		
33 (10 percentile)	19	26
>33	13	18
missing	42	57
Birth Weight for Gestational Age		
≤ 10 percentile	11	15
>10 percentile	42	57
missing	21	28

[*] Postnatal growth deficiency defined as ≤10 percentile for height or weight for age.

[+] Prenatal growth deficiency defined as birth weight for gestational age or birth length ≤10 percentile.

parents' height. These four children all had the cluster of FAS facial features and at least one CNS impairment. These four children were referred from the following sources: private physician (1), early intervention program (1), and parents or legal guardians (2).

Table 3.
FAS[*] Facial Features, Central Nervous System (CNS) Abnormalities, Other Conditions, and Diagnoses Noted Among 74 Clinic Patients

Facial Features	N	%
At least one FAS facial feature noted	34	46
Suggestive of FAS by dysmorphologist	12	16
CNS Abnormalities		
At least one CNS abnormality noted	70	95
Developmental delay (including motor delays)	55	74
Short attention span (includes ADD[+])	51	69
Speech or language delay	48	65
Hyperactivity	38	51
Learning disability	27	36
Short attention span, developmental delay, and speech/language delay	31	42
Other Conditions		
Abuse (among all patients)	40	54
Have at least one of the following:		
Neglect	26	35
Physical abuse	21	28
Sexual abuse	14	19
Behavior problems	31	42
Diagnoses		
Fetal alcohol syndrome (FAS)	4	5
Possible alcohol-related effects, not FAS	51	69
Possible alcohol exposure without effects	19	26

[*] Fetal alcohol syndrome

[+] Attention deficit disorder

The diagnosticians concluded that 51 (69%) of the children may have been affected by prenatal alcohol exposure (Table 3). These 51 children did not have all of the characteristics of FAS, but they did have some characteristics that could be the result of prenatal alcohol exposure, such as growth deficiency or a CNS impairment. Among the 74 alcohol-exposed children, 17 (23%) children who did not receive the diagnosis of FAS had at least one facial feature of FAS, at least one CNS abnormality, and evidence of growth deficiency (≤10 percent). These children were not assigned the diagnosis of FAS because the diagnosticians noted the cluster of facial features suggestive of FAS was not present in 14 children, believed that the growth deficiency for two children was insufficient to warrant the diagnosis of FAS, and assigned a diagnosis of fetal dilantin syndrome to the remaining child. These 51 children were referred from the following sources: Indian Health Service (1), Alaska Native health corporation (1), previous FAS clinic (1), local mental health agency (1), school nurses (2), Head Start (2), private physician (3), public health nurse (3), early intervention program (7), child protective services (11), and parents or legal guardians (19).

Recommendations

The interdisciplinary team developed a care plan that included recommendations to the caretakers of the 74 alcohol-exposed children. The following recommendations addressed needs that had not been previously considered: parents' support group follow-up (38%), referral of the children to a psychologist (35%), further school testing and planning for the children (27%), children's referral to a nutritionist (20%), parents' referral to a parent resource center (19%), medical follow-up (16%), and follow-up with a public health nurse (16%) (Table 4). Some parents requested that a professional staff member come to their homes to assist with their child's behavior management. Other parents also requested respite care (3%). No referrals to an alcohol treatment program were made. At least three recommendations not previously considered were made to parents of 25 (34%) children.

Table 4.
New Recommendations for Parents of 74 Clinic Patients

For child	N	%
Referral to local psychology resources	26	35
Further school testing and planning	20	27
Referral to nutritionist	15	20
Medical follow-up	12	16
Public health nursing follow-up	12	16
For parent		
Parent support group	28	38
Referral to parent resource center	14	19
Referral to financial resources	3	4
Follow-up with respite care	2	3
Referral to alcohol treatment program	0	0
At least one new recommendation	61	82
At least three new recommendations	25	34

Parental Characteristics

Only 26 of the 74 children were living with one or both of their birth parents: 16 children were living with their birth mothers, six children were living with their birth fathers, and four children were living with both birth parents.

More than half of the birth mothers for whom data were obtained were binge drinking (consuming at least 5 drinks per occasion or day) during the pregnancy of the child who was evaluated in the clinic (Table 5). Eighty-three percent of the children living with birth parents were exposed to tobacco in utero. Half of all the mothers reported using alcohol, tobacco, and either cocaine or marijuana during pregnancy. Two (8%) of the mothers were using birth control at the time of their child's conception. Few of the mothers began regular prenatal care during the first trimester (15%) or had been treated for alcohol use before their pregnancy (12%). At the time of their child's clinic evaluation, only 3 (12%) birth mothers stated that they were consuming alcohol, 4 (15%) stated that they were sober, and 19 (73%) refused to answer the question regarding current alcohol use.

DISCUSSION

Population-based diagnostic clinics provide an opportunity to deliver special health services to children affected by alcohol exposure in utero. As earlier studies have indicated, children identified from the clinics who were affected by alcohol exposure in utero and the families of these children required a variety of psychological, medical, social, and educational services (13,14). Because the families tended to focus on the child's psychological and educational needs during the assessment by the interdisciplinary team, most of the clinic recommendations addressed those needs. This focus can be largely attributed to the preponderance of school-aged children referred to the clinic. Long-term cognitive, developmental, psychosocial, and adjustment problems are characteristic of school-aged children with FAS (15-18). Maladaptive behaviors such as poor judgment, attention deficits, and difficulties in interpreting social cues are common among adolescents with FAS (16). Any interdisciplinary team evaluating school-age children suspected of having FAS should include a child psychologist as well as a behavior and education management specialist.

The high prevalence of physical and sexual abuse and neglect among the children evaluated in the FAS clinics was likely due to the fact that a substantial number of these children had been referred by child protective services. The high prevalence demonstrates the need for social service interventions in the homes of many children exposed to alcohol in utero. In a previous study, evidence of physical or sexual abuse was found among 27% of families with an alcoholic parent (19). These data suggest that further studies are needed to determine the impact of physical and sexual abuse and neglect on children with FAS.

We identified several gaps in the referral resources provided to families. Although some parents requested respite care, no respite care was available that specifically addressed the needs of parents of children exposed to alcohol in utero. Other parents requested that a professional staff member come to their homes to assist

Table 5.
Maternal Characteristics for 26 Children Living With Birth Parents

Substance use during pregnancy	N	%	Total N*
Alcohol	26	100	26
Drinks per week			
Median: 18 drinks			
Range: 4-84 drinks			
At least 5 drinks per occasion or day	15	58	26
Throughout pregnancy	16	62	26
Tobacco	20	83	24
Median # of cigarettes per day	20		
Marijuana	13	50	26
Cocaine	12	46	26
Amphetamines	1	4	26
Lysergic acid diethylamide	1	4	26
Heroin	1	4	26
Medications	5	9	26
Alcohol and tobacco with either cocaine or marijuana	13	50	26
Using contraceptive at time of pregnancy			
Yes	2	8	25
Regular prenatal care initiated during first trimester			
Yes	2	15	13
Birth mother treated for alcohol abuse			
Before pregnancy	3	12	26
After pregnancy	15	58	26
Ever treated	1	4	26
Never treated	7	27	26

* Data not available for all children

with management of their child's behavior; however, the clinics did not have enough qualified staff members to provide follow-up home visits to all of these parents. Future FAS diagnostic clinics in Alaska should be modified to address as many needs of parents with alcohol-exposed children as possible.

More than half of the birth mothers who were still living with their children had engaged in high-risk behaviors during the pregnancy of the child evaluated in the FAS clinic. Many birth mothers had been binge drinkers during that pregnancy and could possibly continue to drink during subsequent pregnancies. Yet most birth mothers did not indicate that they were currently using alcohol at the time of the child's evaluation, and none were referred to an alcohol treatment program. Current alcohol use usually was not discussed further with birth mothers at the clinic visit. That alcohol use was not discussed further might be attributed to the focus of the clinic evaluation being more on the child than on the birth mother. In another study, alcohol use did not appear to be a topic usually discussed with pregnant women by health care providers (20). Scheduling a return visit has been recommended as a method to address an alcohol-related problem (21). In addition, clinic staff should be trained to assess a woman's current drinking status and to provide counseling for women who do not report current alcohol use.

In addition to drinking alcohol during pregnancy, many of the birth mothers smoked cigarettes and used illicit drugs during pregnancy. Behavioral Risk Factor Survey results for 1985 through 1988 demonstrated that the prevalence of alcohol use among pregnant women remained highest among smokers (22). Children produced by these mothers are at high-risk for adverse outcomes from the effects of prenatal exposure to tobacco and illicit drugs (23). Intervention efforts should include referrals to smoking cessation and substance abuse treatment programs for mothers who smoke, drink, or use drugs.

Few birth mothers at the FAS diagnostic clinics were using contraceptives at the time of their child's conception. However, information on current contraceptive use was not collected, and no birth mother was referred for family planning. The care planning in the clinic was focused more on the child than on the birth mother. In another study, barriers to women's use of family planning services among patients in drug treatment programs have included not knowing what family planning services were available, believing that they did not need such services, and having a prior negative experience with health care providers (24). In the future, family planning issues might be addressed during a follow-up visit by the public health nurse. Family planning services should be designed so as to be more acceptable to women at high risk of drinking during pregnancy.

Similar to the data collected in previous studies (25,26), data from the State of Alaska clinics suggest that mothers at risk of producing children affected by alcohol exposure in utero did not attend prenatal clinics. Lack of prenatal care is strongly associated with adverse perinatal outcomes including increased risk for low birth weight infants, preterm delivery, and maternal and infant mortality (27). Further studies are needed to determine the reasons for nonreceipt of prenatal care among mothers at high-risk of drinking during pregnancy. If mothers do not seek prenatal care, health care providers miss an excellent opportunity to encourage them to abstain from drinking during their pregnancy (28).

That few previously undiagnosed FAS cases were found through the diagnostic clinics might be the result of children more severely affected by prenatal alcohol exposure having been diagnosed with FAS earlier in their life and already being served by other programs and thus noted by other data sources. Such programs might include the state's early intervention program, child protective services, public health nursing programs, or Medicaid programs. Children with less severe effects of prenatal alcohol exposure might then comprise the bulk of older children seen at the diagnostic clinics conducted by the State of Alaska health department.

Another explanation for the small number of FAS cases found in the diagnostic clinics might be the lack of referrals from private physicians. Previous studies have found that physicians do not routinely ask their pregnant patients about alcohol consumption; the authors of these studies have suggested that physicians might need organized support in developing skills for the early detection and referral of patients at risk for adverse outcomes related to prenatal alcohol use (29-30).

In a survey of health care providers in Alaska during 1992 and 1993, only 63% of pediatricians agreed that making a diagnosis of FAS can improve treatment plans for the affected child (31). In contrast, many more public health nurses (88%), obstetricians (79%), and family practitioners (81%) agreed with the above statement (31). The survey also found that most family practitioners, obstetricians, pediatricians, and public health nurses requested referral resources for children with FAS (31). The State of Alaska clinics should be structured so that physicians are aware that the clinic evaluation includes service referrals provided by the interdisciplinary team.

The data from the State of Alaska clinics suggest that physicians use different criteria to assign the diagnosis of FAS. In our study, one child was diagnosed with FAS even though the child's growth deficiency did not meet the FAS criterion. The use of different FAS case definitions by diagnosticians might prevent meaningful comparisons of results from different studies. To monitor trends and to assess the effectiveness of public health interventions and programs, diagnosticians must use a consistent case definition for FAS.

The finding of only four FAS cases among 74 children required an extensive commitment of time and resources over a four-year period. As part of a surveillance study conducted in Alaska, the 1992-93 data from the clinics were combined with information from the private sector, Alaska Department of Health and Social Services, and Indian Health Service/regional Native health corporation data sources. However, the data from the clinics accounted for only five percent of all FAS cases found in Alaska between 1977 and 1993 (32). The authors of that study suggested that, if linked, the FAS-related referral and diagnostic clinics described here, early intervention programs, and Medicaid would be useful as data sources for statewide FAS surveillance (32). An earlier study had also recommended linking multiple data sources for FAS surveillance (33). Rates based on these data sources would provide a minimum estimate of FAS prevalence in a state (32).

Unlike the other Alaska Department of Health and Social Services data sources examined in the previous FAS surveillance study conducted in Alaska (32), the diagnostic clinics provided a method for active FAS case-finding. Health care providers actively searched their case files for possible cases of FAS. Children identified as possibly having FAS were evaluated for FAS in the diagnostic clinics, and their families were referred for special health services. The other Alaska Department of Health and Social Services data sources in the Alaska FAS surveillance study utilized a passive case-finding methodology (34). These data sources were not designed to ascertain cases of FAS. The data sources found FAS cases by reviewing the child's existing records and did not rely on an evaluation of the child by experts in FAS. A study conducted in Colorado suggested that FAS surveillance can be accomplished by linking active case-finding with an existing birth defects registry system (35).

Further studies are necessary to improve the delivery and evaluation of services through FAS diagnostic clinics both to the children affected by alcohol exposure in utero and to their families. Because birth parents will attend FAS diagnostic clinics with their children, the interdisciplinary team should focus on providing the appropriate services both to the children with prenatal alcohol exposure and to their birth mothers. Further, the FAS diagnostic clinics provide a method of linking the delivery of special health services to children exposed to alcohol in utero with FAS surveillance. Statewide FAS surveillance can be accomplished by linking FAS diagnostic clinics with other state data sources. FAS surveillance can provide information to identify at-risk populations and to guide, plan, and evaluate FAS prevention efforts.

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Alaska's State Public Health Laboratories (ASPHL): A Crisis Situation

Gregory V Hayes, Dr PH, HCLD (ABB)(1)

ROLE OF ALASKA'S PUBLIC HEALTH SYSTEM

The health of individuals begins with a safe and healthy community in a safe and healthy environment (9). Alaska's unique environment, demographics, and seasonal tourist industry continually challenges our public health system. Alaska needs to retain a strong state public health system that is based on prevention and extends well beyond individual medical care to the entire population (9). Keep in mind our biggest gains in health status have come from population based programs targeting sanitation, safe drinking water, tobacco prevention, immunization, and the control of infectious diseases (3).

Misperceptions about public health are common among the general population. One of the most common misperceptions is that public health's major role is to provide state-subsidized health care to the poor and indigent. Clearly this is not public health's major role. Public health, is based on the premise that the economic, social, and cultural development of any community is dependent on the health and safety of its citizens. It is the commitment by this State at every level, to assure safe and healthy communities to all citizens, so that Alaskans can devote their energies to fulfilling their dreams. This is a responsibility of the State which cannot be delegated, even in part (1,4). This mission is addressed in Alaska's Constitution, Article VII, Section 4: ". . . the legislature shall provide for the promotion and protection of public health."

The essential activities of the public health system are assessment, policy development, and assurance (Table 1) (2,4,6). It is important to note that Alaska's State Public Health Laboratory (ASPHL) is critical to each one of these activities (4).

Table 1 (4)

Assessment
- Assess the health needs of the community.
- Investigate the occurrence of health effects and health hazards in the community.
- Analyze the determinants of identified health needs.

Policy Development
- Advocate for public health, build constituencies and identify resources in the community.
- Set priorities among health needs.
- Develop plans and policies to address priority health needs.

Assurance
- Manage resources and develop organizational structure.
- Implement programs.
- Evaluate programs and provide quality assurance.
- Inform and educate the public.

ROLE OF ALASKA'S PUBLIC HEALTH LABORATORIES

The ASPHL is an essential component of the State and National public health infrastructure and clearly operates with a different mission and purpose than do private sector laboratories. Nor do they perform identical types of testing when compared to clinical laboratories. The ASPHL provides a broad range of disease control, preventive health care, environmental monitoring, applied research and laboratory improvement services. They generate information that serves the needs of epidemiologists, regulators, policy makers, the medical community and the public, the ultimate beneficiary. State public health programs, depend on the high quality data which is produced by the ASPHL. Aside from daily services performed to support State programs, the ASPHL maintains the expertise and flexibility to investigate outbreaks, conduct surveillance activities and provide support as part of the State's disaster preparedness plan for response to emergencies. The reemergence of infectious diseases and drug resistance, combined with the increase in interstate and international travel make the need for an effective national public health laboratory network greater today than ever before. The ASPHL represents a first line of defense for Alaskans in the rapid recognition and prevention of communicable diseases and is a critical component of the national network (5,8).

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(1) Gregory V. Hayes, Dr.P.H., HCLD (ABB)
Chief, Section of Laboratories
Alaska Dept. of Health & Social Services

Integral cooperation exists between the public health laboratory and the State's epidemiologists in investigations, surveillance, surveys and control activities. The laboratory performs tests that are clearly not a part of individual medical care such as the testing of contacts or family members. Frequently there is a need to determine the health status of well persons through screening studies (10). Such testing is essential in the control of diseases. Tests performed in the ASPHL comprise disease assessment, which help protect the community by preventing and controlling disease. Tests performed are done without compensation as the motive but as "advocacy for the public's health". The cost of the test in the public health laboratory pays for more than a test result. It provides health benefits to the local population and the nation as a whole (4). The ASPHL performs population based screening for conditions of public health concern such as human immunodeficiency virus and sexually transmitted diseases (STD) such as gonorrhea, chlamydia, and syphilis. The ASPHL also provides specialized testing for low-incidence, high-risk diseases such as tuberculosis and rabies. Testing performed for disease agents such as protozoa, enteric bacteria, meningococci, STD, influenzae viruses and other respiratory diseases is focused on determing epidemiologic shifts and newly emerging pathogens (9). This is accomplished through analysis of the lab's own primary isolates and those referred to them by private laboratories throughout the state. Epidemiologists use the APHL's data to determine "sentinel events" that would indicate an emerging problem (9). Examples of this include hantavirus, antibiotic-resistant mycobacteria, Escherichia coli 0157:H7 and cryptosporidiosis. Additionally the ASPHL provides specialized reference services for other laboratories in the State such as antibiotic susceptibility testing and typing of isolates contributing not only to patient care but providing tools for disease surveillance. The ASPHL also tests foods associated with outbreak investigations which is clearly not a procedure undertaken by clinical laboratories. Another major responsibility of the ASPHL is laboratory improvement within the State. This is accomplished by providing training, technical assistance, and proficiency testing to improve the quality of work performed by other laboratories in the state. Lastly, the ASPHL conducts applied research to develop or evaluate new laboratory methods in order to aid epidemiologic investigations or for assessment of public health services (9). Unlike many states, the ASPHL do not provide newborn screening, blood-lead tests, toxicology screens, or significant environmental testing.

BACKGROUND AND CURRENT STATUS OF THE STATE'S PUBLIC HEALTH LABORATORIES

There has been a Public Health Laboratory in Alaska since the late 1930s when the first facility was established in Juneau. Two other laboratories, in Anchorage and Fairbanks, were established in 1939 and 1948 respectively as the populations grew. The primary purpose of these facilities at the time was to provide direct clinical support to local physicians and analytical support to sanitarians, veterinarians, and other environmental professionals.

Today, all three laboratories operate under the State Division of Public Health. Each lab represents geographically diverse and distant populations, but populations that share a much greater accessibility than when the ASPHL were first established. Since 1985, thirteen studies have accessed the deficiencies and deteriorating state of the ASPHL. The buildings that house the three laboratories are in leased space that is technically or functionally inadequate to support the types of testing conducted there. Two of the buildings (Anchorage and Juneau) are in urgent need of repair and are unable to meet the demands of changing analytical technologies. In addition, the Anchorage and Juneau buildings, which were originally designed as office buildings, face serious mechanical system and structural inadequacies for conducting laboratory testing and analysis. The Fairbanks laboratory is housed in a dated facility on the University of Alaska campus. All of the studies document significant health and safety risks caused by improper ventilation and poor layout of lab spaces. In addition, lease expenses and the increasing cost of renovating leased space to meet minimal safety standards continually strain the resources of the Department of Health and Social Services. Serious risks to the State exist if the status quo of the ASPHL continues (7).

The following issues affect the cost-effectiveness and level of service of the ASPHL (7):

- Health and safety risks caused by improper ventilation and other facility limitations.
- Leased facilities with limited maintenance or repair resources.
- Duplication and redundancy in facilities.
- Limited space for growth or addition of new technologies.
- Procedural inefficiencies caused by poor facility layouts.
- Duplication and redundancy in staffing.
- Duplication and redundancy in administrative tasks.
- Unwieldy and time consuming transfer of specimens between sites.

- Reduced ability to communicate, coordinate, and train; duplicated supervision; diminished team-building capabilities.
- Slow turnaround time for test results.

FUTURE OF THE STATE'S PUBLIC HEALTH LABORATORIES

As emphasis on prevention increases, so will the need for preventive services, it is not possible to know what research will bring to the health care system and to disease prevention in the future; also it is not possible to predict the services that public health laboratories will need to provide. Microorganisms continue to evolve and sometimes offer surprises. The ASPHL will be expected to detect promptly and confirm an ever-expanded array of pathogens as they are discovered in the future. HIV will not be the last infectious disease. There will continue to be other new, as well as re-emerging, infectious diseases in the United States, reflecting new discoveries (pneumonia due to chlamydia) and changes in the environment (Lyme disease), technology (Legionnaires Disease), personal behavior (herpes 2, human papilloma virus, and other sexually transmitted diseases), age of population (pneumonia, influenza), food production practices (salmonella, small particle viruses), poverty (tuberculosis), and the microbial world (antibiotic resistance, virulence). Increased travel, immigration, and translocation of populations increasingly expose citizens to malaria, dengue, parasitic diseases, hemorrhagic fevers, and enteric diseases, such as cholera. In all these instances, the first point of contact and often the only source of expertise is the public health laboratory (4).

It is obvious that public health laboratory services are essential for the proper functioning of the public health system in the State of Alaska. There will continue to be a need for core public health functions and for expertise of personnel trained in public health laboratory work. Private laboratories cannot be expected to provide equivalent services (4).

Smart management dictates that a state public health laboratory meet the standards of economic and administrative efficiency while providing a safe and efficient public health service. Those standards can no longer be met through the independent operation of three separate laboratories operating in facilities that have outgrown their utility (7). Without adequate facilities, space and equipment, the types and volume of tests offered by the laboratory are reduced, the remaining staff are overworked and overcrowded, morale drops, user confidence erodes, and the downward spiral of the laboratories' capabilities, reputation, and public health relevance accelerates. This is hardly a recipe for success (4).

It is crucial for Alaska to have a public health laboratory that will gain the confidence of the medical profession and our citizens while assuring that highly technical and skilled staff can be recruited and appropriately supported in meeting all present and future demands.

A new facility to house the ASPHL is desperately needed in order for the laboratory to continue meeting the goals and objectives of their public health mission. Our major concern is in keeping intact, and indeed strengthening, the public health laboratory system that is so crucial for carrying out the basic functions of public health in Alaska(3).

The future of the public health laboratory depends heavily on the ability of health department personnel, appointed and elected officials, the medical community and the general public to understand and appreciate the meaning of public health and the functions of the public health laboratory(4). Those who believe in the role of disease assessment and disease prevention need to advocate for a new Public Health Laboratory facility that pays for itself, integrates and adequately houses departmental laboratory functions, allows for future growth and provides for a satisfactory level of services at a lower cost (4).

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(continued on page 147)

American Society for Circumpolar Health

PROGRESS IN CIRCUMPOLAR HEALTH

The American Society for Circumpolar Health (ASCH) is now entering its thirtieth year, since its founding by Drs. Earl Albrect and Fred Milan and other Alaskan scientists in Fairbanks at the First International Symposium for Circumpolar Health at the University of Alaska Fairbanks, July 23-28, 1967. While our numbers remain small (119 paid-up members in good standing, with 7 new members in 1996), we have played a key role in the circumpolar health movement, and will continue to do so.

NEWS

The Tenth International Congress on Circumpolar Health (XICCH), held in Anchorage this May, was a resounding success. Over 700 registrants from 15 nations attended the consistently excellent plenaries, symposia, oral and poster sessions, and special programs. In addition to featuring some very prominent scientists and public health workers of world renown, the program afforded many excellent opportunities for international interaction by those of us working in public health and basic science research. The indigenous program, integral to the entire Congress this year, was also a resounding success. All of those volunteering the many hours necessary to successfully organize this major event are to be thanked and congratulated by all of us who benefited from the result. The Proceedings volume for the Congress is now in preparation, and should provide a very helpful reference and archive for the future. While copies will be sent to all full registrants to the Congress, additional copies will be available for sale to those unable to attend.

Following up on the successful program jointly assembled in January, 1995, we have agreed to co-sponsor, with the International Union for Circumpolar Health (IUCH) and the Institute for Circumpolar Studies at UAA, semi-annual Circumpolar Public Health Symposia. The inaugural Fall1996 Symposium will be held November 1 from 1:00 to 4:30 PM in the UAA Business Education Building, Room 207, and includes a highly interesting and varied collection of papers and presenters. If this journal reaches you after that date, do not despair, as we are already planning the Spring program.

Dr. Neil Murphy, ASCH Vice President, chaired a general meeting at the American Association for the Advancement of Science (AAAS) Arctic Division Symposium in Girdwood on September 20. Some highpoints of Dr. Murphy's report: The meeting was well attended and provided a springboard for planning the ASCH's Spring 1997 Symposium [see below]. The focus of a brainstorming session at that meeting was:

- Development of increased Alaska Native participation in science and health care: What has worked, what hasn't worked, and how to improve. Possible resources include: the Della Keats Program, Alaska Rural Systems Program, and WAMI, as well as a number of prominent Alaska Native leaders discussed at the meeting (who will be contacted about this in the near future).

The Spring Conference will be coordinated by Dr. John Booker, Circumpolar Institute, UAA, and Patricia Cochran, of the Alaska Native Science Commission. Please contact them (or me) if you wish to be involved in planning or as a participant.

The annual (official) meeting of the ASCH will be held on Tuesday, December 3, at 12 noon, at the Sheraton Hotel (room to be announced) in conjunction with the 1996 Alaska Health Summit. We will be planning for additional future activities, considering some legislative resolutions (see below), and installing two newly-elected board members (General Directors). Current and prospective members are welcome, and we will conduct the meeting efficiently, to permit those of us who wish to dine together at one of the local bistros to do so after the meeting.

LEGISLATION

An important part of good citizenry and public health practice is involvement in the legislative process. While the last session of the Alaska Legislature did result in some helpful new legislation (please see the Alaska Public Health Association's [ALPHA] Legislative Roundup in the June 1996 ALPHA Newsletter), there remain three very important pieces of legislation requiring attention during the next session: 1) the bill to increase the state's tobacco tax, an important measure toward reducing adolescent smoking that was thwarted by a series of procedural maneuvers by its opponents. Thus, it never came to a floor vote in the House, despite widespread public support and inclusion by Governor Knowles in the Special Session agenda; 2) a bill to authorize the construction of a new state public health laboratory (please see Dr. Gregory Hayes' article on this subject elsewhere in this issue of *Alaska Medicine*), a very important move toward a stronger state public health infrastructure, and 3) the Alaska Health Data Bill, which would greatly improve our disease and injury surveillance infrastructure, and enable much more accurate epidemiologic analysis and response, public health planning, and more efficient use of resources in our state. I urge you to become involved in the legislative process, by discussing these proposals with colleagues and friends, considering resolutions within the ASCH, ALPHA, and ASMA, and personally contacting your legislators with your opinions.

FUTURE OPPORTUNITIES

Much of the ASCH's involvement in international circumpolar health is via its collaboration with the IUCH, of which the ASCH is one of the adhering organizations. The presence of three ASCH representatives (Dr. Grace Egeland, Patricia Cochran, and myself) on the newly-elected IUCH governing council, as well as my service as IUCH President (1996-1999), affords a new level of involvement and potential influence over the IUCH by the ASCH membership. In addition to the triennial congresses, the IUCH consists of a growing number of working groups, currently including: 1) Cancer; 2) Diet and Heart; 3) Environmental Health; 4) Family Health; 5) Fetal Alcohol Syndrome; 6) Health Interview Surveys; 7) HIV, AIDS, and STDs; 8) Injury Prevention; 9) Occupational Safety and Health; 10) Tobacco and Health; and 11) Women's Health. The central focus of future efforts of the IUCH will be on utilizing the working groups as structures to enable collaborative research and public health interventions. The IUCH has been fortunate to recently receive a U.S. Centers for Disease Control and Prevention (CDC) Conference Grant. As the $10,000.00 so far awarded originated from the National Institute for Occupational Safety and Health, we intend to apply these funds toward the support of the teleconferencing and internetworking capacity of the IUCH Injury Prevention and Occupational Safety and Health Working Groups. As funds become available, we will extend the following functions to all of the IUCH working groups.

Toward that end, during the remainder of this year, we plan to modify electronic groupmail software to enable asynchronous e-mail communication among Working Group members worldwide. We are currently investigating available options for low-cost teleconferencing using the Internet and acquiring and pilot testing Internet teleconferencing hardware/software. Our first real-time interaction for working groups via Internet ("chatgroup") will occur in late 1996 or early 1997. We plan to pilot test Internet-based videoconferencing between Anchorage and at least one Nordic Nation site, then hold additional working group meetings by telephone or Internet audioconferencing, with collaborative data-gathering and/or -sharing. By early Spring, 1997, we plan to hold formal (i.e., strict agenda, scheduling, speakers/presentations) teleconferences for each working group, in a microcomputer-based hypermedia environment. We also plan to televise the Spring 1997 collaborative IUCH/American Society for Circumpolar Health/Circumpolar Health Institute Public Health Symposium via the Internet to Nordic nation, Greenland, Canadian, and Russian IUCH collaborating centers. Lastly, we will be exploring the utility and practicality of technology and skill transfer to underserved Circumpolar areas with great need (e.g., the Barents Region of Northwestern Russia).

Given the many common goals shared by the IUCH and ASCH, I would like to extend an invitation for ASCH members to join and play an active role in IUCH working groups, as well as to get involved in seeking extramural funding for the working group and Internet capabilities.

In the past, the IUCH journal, *Arctic Medical Research,* has provided a good means for periodic communication among members. However, the future existence of that journal is in jeopardy since the funding of the Nordic Council for Arctic Medical Research (NoCAMR) will terminate by December 31st, 1996. One of the two editors (Dr. Hans Åkerblom) wishes to step down as editor by the end of 1996 while Dr. Juhani Lepäluoto has agreed to continue. A grant from the University of Oulu (Finland) has secured sufficient financial support for 1997 and from next year on, the Nordic Society for Arctic Medicine will take over the joint responsibility for the journal together with IUCH instead of NoCAMR. A working group has been appointed to look into the future of the journal. The working group consists of Drs. T. Kue Young (Canada, immediate past President of the IUCH), Juhani Leppäluoto (Finland), and Peter Bjerregaard (Denmark).

The major collaborative project being considered currently by the IUCH, ASCH, and the other adhering organizations is a white paper or book (dependent on funding) on health status throughout the circumpolar region. This volume will provide a valuable resource for planning for the region, as well as give a clearer identity to some of the cross-cutting issues across the region (environmental contamination, subsistence lifestyles, indigenous health and self-determination, community-originated research, emerging infections, injuries). Volunteers are needed to assist in acquiring and compiling vital and morbid statistics for the circumpolar region for this important effort. Please contact me if you are interested.

I look forward to seeing current and potential members on December 3.

George A. Conway, M.D., M.P.H.
President, American Society for Circumpolar Health
e-mail: goc1@NIOSR2. em.cdc.gov

Telephone Advice: Is it a Service or a Hazard?

Thoughtful Ways to Communicate Bad News

"This is Nancy Grant, Bobby's mother. He's had a fever and runny nose for the past two days and we were planning to leave on vacation tomorrow. Can Dr. Smith give Bobby something for his cold?"

"Hi, Fred Watson calling. The medicine Dr. Jones gave me after my surgery yesterday is making me nauseous. What should I do?"

"I'm in my sixth month [of pregnancy] and I've been spotting a lot lately. Is that normal?"

These questions are typical of the inquires many medical offices receive daily from patients who seek advice, treatment recommendations, prescriptions or reassurance from their doctor. The volume of calls tends to increase in bad weather periods, especially during the "cold and flu season," when people are less willing to venture outdoors. Requests for telephone advice are likely to increase as more patients enroll in managed care plans that discourage unnecessary office visits or won't pay for visits to an emergency department that, retrospectively, the plan deems were not emergencies. For many patients, the expense of an office visit is their incentive to want to resolve medical problems with a phone call.

> **"Giving medical advice on the telephone may be a convenience for patients... but it can be a costly practice for the doctor who tries to assist a patient without the benefit of a physical exam."**

Some doctors welcome and encourage phone calls in lieu of a visit, but others find they are unable to handle the growing number of calls, or worry about malpractice liability if they are sued for misdiagnosing a condition reported by phone. "Giving medical advice on the telephone may be a convenience for patients," says a malpractice defense attorney, "but it can be a costly practice for the doctor who tries to assist a patient without the benefit of a physical exam. Doctors say that the art of practicing medicine often entails skilled observation. Sometimes a doctor can *see* that a patient is sick, before an exam or tests prove it. Diagnosing is more difficult if the physician cannot observe the patient directly."

Physicians who dispense telephone advice believe the practice reduces unnecessary visits for minor problems that respond to first aid or basic care, such as taking aspirin, elevating feet, staying in bed, trying an over-the-counter medication, or taking a warm bath. Others add that giving phone advice promotes better patient relations. "If patients perceive their problem is a minor, although irritating one," says a family physician, "they resent having to make a appointment or take time off from work to come in, especially if during an office visit they find the doctor's advice could have been given on the phone."

Many clinics and high-volume practices such as pediatric offices have a formal telephone advice program, usually staffed by a trained nurse who can screen (triage) calls and make judgments about which of them require the doctor's immediate or prompt attention. But triaging by telephone has its limitations, says Maryann Hauf, RN, a former telephone advice nurse for a large health maintenance organization that encourages patients to call before they make an appointment with a doctor. "Two of our biggest concerns were deciding how serious a patient's symptoms were, and eliciting enough meaningful information from ill patients to make a decision," Hauf says. "Some people who are quite sick and should be seen by a physician keep putting off a visit, hoping a phone call will save them the trip. Advice nurses must err on the side of caution and advise patients to come in if there is any question about the seriousness of their complaints."

Pros and cons of phone advice

Proponents say phone advice programs offer these benefits:

✔ Minor illnesses can be managed without the expense of an office visit;

✔ often the patient is experiencing a recurrent problem that can be treated with medication that worked well in the past;

✓ costs of office visits are reduced, a plus in capitated managed care plans;

✓ a doctor's willingness to manage minor problems by phone promotes good will.

Some of the liability risks of dispensing advice by telephone:

☐ A physician is not able to personally assess the patient's appearance, severity of symptoms, body language or other factors the doctor would take into account while doing a physical exam;

☐ patients - or others calling on their behalf - may be unreliable historians or give an inaccurate assessment of the severity or location of physical complaints; lay people often incorrectly describe or minimize symptoms, a potentially serious omission in impending heart attack cases;

☐ unclearly-worded questions may elicit misleading responses. In a pediatric malpractice case that resulted in settlement for a misdiagnosis of meningitis, the doctor had asked a patient's mother who called for advice if her child "has a fever;" the mother responded in the negative. The doctor later learned that the mother did not have a thermometer, but had assumed the child was afebrile because his forehead did not seem warm to touch. Given the child's other reported symptoms, the doctor conceded, it would have been important to know the patient's actual temperature;

☐ increased volume of phone calls may strain the phone system or staff, although adding a phone line and encouraging patients to limit non-emergency calls to specific times might alleviate these burdens.

Reducing liability risks

These steps can minimize the liability risks for physicians and clinics who desire to dispense medical advice by telephone:

- Permit only physicians or qualified staff (e.g., nurses, physician assistants) to handle telephone advice calls;

- use phone advice guidelines that limit the types of situations non-physicians can handle by phone; guidelines also should list questions to ask callers and instructions for actions the phone adviser should take in specific situations; the Loss Prevention Department at Medical Insurance Exchange of California, a physician-owned liability insurer, has sample phone advice guidelines physicians can adapt; for copies write: MIEC Loss Prevention, 6250 Claremont Avenue, Oakland, CA 94618;

- inform patients in writing about proper use of this telephone service; explain the types of complaints the doctor and staff usually can handle by phone and those that might require an office visit;

- advise patients each time they call about the limitations of dispensing medical advice by phone; tell patients how soon they should call back if their condition does not improve or if it worsens;

- give callers ample time to explain their problem; avoid leading questions that can result in less-than-accurate responses. For example, rather than ask "Have you had this pain for a long time?" ask "How long have you had this pain?" Instead of "Do you have any chest pain?" ask "Exactly where are you having pain?" Ask the caller to describe the type and severity of the pain;

- physicians and aides who dispense phone advice should avoid using medical terms or acronyms; lay people may not know that common words (e.g., "void") can have a separate, medical meaning;

- when in doubt about a caller's accuracy or reliability, encourage an office visit; pay attention to your professional instincts;

- carefully evaluate a patient's contention that the current problem is identical to a similar complaint the doctor treated earlier;

- physicians should be cautious about prescribing medication by phone for a new complaint; a wrong diagnosis can result in an ineffective or harmful prescription;

- document all phone advice calls; use the caller's words, when possible; it is preferable to document all calls in each patient's chart, but for uncomplicated, routine calls, a phone log may suffice. Physicians who permit staff to handle advice calls should review their documentation to ensure phone advice guidelines were followed, appropriate advice was dispensed and the physician was properly informed of calls in individual cases.

> Advise patients each time they call about the limitations of dispensing medical advice by phone. . . [and] how soon they should call back if their condition does not improve or if it worsens.

Experts' advice for giving bad news

No one likes to be the bearer of bad news. But it often is difficult for physicians to avoid this obligation. The bad news may be a positive test result that indicates further tests or treatment are needed, or identifies a terminal diagnosis. Disclosing bad news to a patient on the telephone can be traumatic and insensitive. Jeanne Miranda, PhD, and Robert V. Brody, MD, in the *Western Journal of Medicine,* say that some medical schools now teach professionals how best to convey bad news to patients and families. "Bad news can involve a variety of things," the authors note, including, "a positive test for the human immunodeficiency virus, a heart attack, death of a family member, a recommendation for a surgical procedure, loss of a pregnancy, the need for a prolonged hospital stay. Anything that changes a person's view of the future in a negative way is bad news. . . The physician should remember that bad news, like beauty, may be 'in the eye of the beholder'." [Communicating bad news, *West J Med 1992* Jan; 156:83-85]

When and where bad news is disclosed makes a difference in how the person receiving the news reacts. Learning of bad news when one is alone can cause anxiety, fear and resentment toward the messenger. The telephone can be a unintended barrier between the patient and physician in such situations, limiting the doctor's ability to use eye contact, silent pauses, body language and hand-holding to comfort a patient or family member. Among the "Principles of Communicating Bad News" Drs. Miranda and Brody offer in their article:

- Give the news in person; avoid giving bad news by phone. *[Miranda and Brody note that one study found that 76% of patients who were told bad news in an office or hospital "rated their physicians as 'helpful' in aiding them to understand their illness, while only 48% of those told on the telephone or in the recovery room rated their physicians in this way."]*

- Choose a quiet setting where you won't be interrupted.

- Assess the patient's physiologic and emotional state; *[it may be prudent to delay the discussion if the patient or family members are overly distraught].*

- Use clear, simple language.

- If an interpreter is needed for a discussion with the patient or family, talk to her or him in advance, to make sure the interpreter understands the clinical situation and the goals of the discussion.

- Express sorrow for the patient's pain; be human.

- Be realistic; avoid the temptation to minimize the problem, but don't take away all hope.

- Explore how the patient feels after receiving the news. Offer to answer the patient's (or family's) questions.

In hospitals, social workers, clergy and patient representatives can assist physicians or nurses to communicate bad news, particularly when a death is involved or if arrangements for follow-up care must be made. Their skill and experience in comforting patients at such times are invaluable assets.

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Sexually Speaking . . .

Mary Cavalier, M.S.(1)

FROM LOVER TO CAREGIVER . . .

Pharmacologically-induced Sexual Dysfunction

One may wonder how a chemotherapy patient experiencing all the side effects thereof can even consider being sexual? I have heard patients talk about a need to feel a sexual connection with their partners. "My body is betraying me. At least, for a moment, I can feel good." Sexual experiences can be an escape from the reality of the disease — for both people. Therefore, to combine pharmacologic sexual dysfunction adds insult to injury.

The following are reported drug-induced sexual dysfunction from the most commonly used chemotherapy agents (Micromedex, Inc., 1974-1996)

- DECREASED LIBIDO
  - CNS depressant
  - antiandrogenics
  - chlorpromazine
  - flutamide

- ANORGASMIA
  - fluoxetine

- IMPOTENCY
  - chlorpromazine
  - flutamide

- DELAYED ORGASM
  - fluoxetine

- INHIBITED EJACULATION
  - fluoxetine

- DECREASED SPERM PRODUCTION
  - cyclophosphamide

The good news is that most of the side effects do stop after treatment has ended (Micromedex, 1974-1996). As with any medication, the physician should brief the patient on potential side effects. When it comes to sexual dysfunction and the potential harm to self esteem, education is the best preventative medicine.

In the next issue we shall discuss the psychological effects of treatment.

---

(1) Mary B. Cavalier, M.Ed., Counselor, Educator, Private Practice.

Letter to the Editor . . .

Dear Editor:

Though it is not traditional for the journal to publish obituaries about a member's spouse, I felt that a publication of the obituary of Grace Akiyama might be of interest. She was a remarkable person and her husband, Dr. Henry Akiyama, is an exceptional physician and person as well. The Akiyamas are so well known to so many people here in Juneau that the funeral service held at Centennial Hall was filled with several hundred people.

The memorial remarks and reminiscences were led by Dr. Akiyama himself and were inspiring. Dr. Akiyama gave a touching, warm, and often humorous and light-hearted tribute to her and about their life together, which was appreciated by all in the audience. He graciously set a light tone which cheered a somber, though beautifully executed, memorial service.

Kenneth W. Moss, MD

Obituary

Grace Akiyama

Grace Akiyama, a longtime Juneau resident and community volunteer, died August 24, 1996 at the University of Washington Medical Center in Seattle, Washington, shortly after being diagnosed with T-cell lymphoma.

Grace Taeko Ebihara Akiyama was the first born to Taneyoshi Ebihara and Toye Ijuin on February 24, 1927 in Portland, Oregon. Her mother was the direct descendent of the third son of the Daimyo, Lord of Satsuma. Her father was the elder son of the Shinto priest and guardian of the Grotto, where Amaterasu-Omikani, the first Emperor was to have descended from heaven. The Ebiharas were also of Shizoku class. They were from Kagoshima, Japan.

Akiyama attended Shattuck Grade School and Lincoln High School, both now a part of Portland State University. She also attended Nampo Gakucu, one of two Japanese language schools in Portland. She studied piano at Ellison-White conservatory, later known as Portland School of Music.

In May, 1942, 120,000 Japanese and Japanese-Americans were relocated from the West Coast to 12 relocation-internment camps during World War II. Those from Portland were interned at the temporary Portland International Exposition Stockyards, called "Assembly Center." They were then moved to a semipermanent facility called Minidoka Relocation Center, where they resided until the war ended in 1945.

She graduated from Hunt High School in Minidoka and received special permission to attend Ohio Wesleyan University in Delaware, Ohio. When the camps closed in 1945 and her family moved back to Portland, she transferred to Reed College, where she received a bachelor's of science degree in biology.

She married Dr. Henry I. Akiyama on September 7, 1952. The couple and their two children moved to Juneau on June 26, 1961.

She was a lifelong member of Portland's Methodist Church. Upon moving to Juneau the family transferred to the Juneau Methodist Church, now Northern Lights United Church, where she was an active member. Akiyama was a delegate to the Provisional Japanese Methodist Conference and to the Alaska Methodist Church Conferences.

She served a six-year board post with the YWCA in Portland, Oregon. Akiyama was a past member of the Juneau Garden Club; St. Ann's Hospital Guild; a 17-year member, officer and president for four years with the American Lung Association; she served three years as the Alaska representative on the National American Lung Association Board; and Zach Gordon Teen Club Board. She was currently on the Salvation Army Board and Auxiliary; Pioneers of Alaska Auxiliary Igloo No. 6, PEO Chapter G and was a member of the Friends of the Museum.

Akiyama was a talented flower arranger; she studied Shibui, preferring the classical style (Misho School for Flower Arranging). She enjoyed cooking and baking and was a seamstress who sewed all the men's vests and several girls' dance costumes for the first Hoochinoo and Hot Cakes Production. She also created several pieces of needlework for Dr. Akiyama's office, particularly during the holidays.

In celebration of Japan's Girls' Day every March, she displayed her Japanese doll collection and opened her home to local children for a cross-cultural experience.

(continued to page 147)

MY UNCLE, A DOCTOR

MY UNCLE WAS A GENERAL PRACTITIONER IN AN EAST TEXAS TOWN. HE WAS AMAZINGLY VERSATILE AND ALWAYS BUSY. HE LIKED OBSTETRICS AND GYNECOLOGY BEST BUT TOOK ALL-COMERS FROM INFANTS TO CENTENARIANS. HE WAS A FINE SURGEON, AS ADEPT AT MASTOIDECTOMY AS PROSTATECTOMY.

MY UNCLE WAS TACITURN, SOMETIMES EVEN GRUFF—AT LEAST TO HIS NIECES AND NEPHEWS. WE LOVED HIM DEARLY. HE SUFFERED ME MORE THAN MY BROTHERS AND SISTER, I LIKE TO THINK, BECAUSE I WAS INCLINED TOWARD BECOMING A DOCTOR WHEN I GREW UP.

ONE CHRISTMAS SEASON LONG AGO – IT WAS IN THE LATE 1930S –MY FAMILY DROVE FROM ANOTHER STATE TO TEXAS TO BE WITH OUR RELATIVES, INCLUDING MY UNCLE AND HIS WIFE, MY AUNT. ALL WERE TO GATHER IN THE STATELY OLD FRAME FAMILY HOME ON CHRISTMAS EVE.

LATE ON THAT STORMY AFTERNOON, MY UNCLE FINISHED HIS OFFICE HOURS AND STOPPED BY THE HOUSE TO ASK ME IF I WANTED TO GO WITH HIM ON A HOUSE CALL FORTY MILES AWAY IN THE COUNTRY. OF COURSE, I DID! HE PROMISED EVERYONE THAT WE WOULD BE BACK IN TIME FOR SUPPER, KNOWING FULL WELL, I SUSPECT, THAT WE WOULD NOT.

OFF WE WENT IN SWEEPING RAIN. SLICK PAVED ROADS GAVE WAY TO EVEN SLICKER RED CLAY BYWAYS DEEP IN THE PINE AND HARDWOOD FORESTS OF THE COUNTRY. MY UNCLE SAID LITTLE, TELLING ME ONLY THAT HIS PATIENT, A WOMAN, HAD CANCER. I NOTICED THAT HE HAD BROUGHT A BOX.

IT WAS NEARLY DARK WHEN WE STOPPED AT AN UNPAINTED DWELLING, UP ON POSTS, FLOOR SAGGING, WITH A WIDE CENTRAL BREEZEWAY -“A DOG-RUN HOUSE” TYPICAL OF EAST TEXAS IN THOSE DAYS. THE PATIENT’S HUSBAND AND SOME OTHERS GREETED THE DOCTOR AND SAID, “COME ON IN.” UNCLE LANGSTON ASKED ME TO COME IN TOO.

THERE IN THE CENTER OF A LARGE BED LAY A GAUNT WOMAN, ILLUMINATED FROM THE SIDE BY A KEROSENE LAMP ON A TABLE. SHE MURMURED WEAKLY, “OH, DOCTOR NELSON, THANK YOU SO MUCH FOR COMING.”

HE LAID THE BOX ON THE BEDCLOTHES. IT WAS CANDY. HE KNEW THAT SHE COULD NOT EAT IT.

THE DOCTOR SAT BY THE BED AND TALKED IN LOW TONES TO HIS PATIENT FOR SEVERAL MINUTES. THE HUSBAND WALKED US BACK TO THE CAR. WE DROVE AWAY.

TO THIS DAY, I HAVE WITNESSED FEW PROFESSIONAL ACTS TO MATCH THE SOLICITUDE OF MY UNCLE FOR HIS PATIENT ON THAT SOMBER CHRISTMAS EVE NEARLY SIXTY YEARS AGO.

RODMAN WILSON, MD

GLIMPSES OF ALASKAN MEDICAL HISTORY

Edited by Robert Fortuine, M.D.

A shamanistic healing ceremony on the Kuskokwim (1842-1844)

Lieutenant Zagoskin's account of his travels is invaluable as one of the first to describe in careful detail some of the ethnographic features of the peoples of Southwestern Alaska. In the segment to follow, he reports on what was apparently an eyewitness account of a healing ceremony carried out in a *kazhim*, or "men's house" among the coastal Eskimos.

Several features of this story are notable. Although the *kazhim* was normally limited to the men of the village, the principal shaman here is a woman, who is actually assisted by several males, who sing, chant, or beat on drums. A typical feature of such healing ceremonies (paralleled in many other cultures of the world), was the eerie setting, dimly lighted by flickering lamps, and a hypnotic "atmosphere," created by the use of beating drums, chanting, and the sounds of animals. The spectators sat huddled close to one another on benches arranged along the walls. The shaman herself seems tentative, at first, searching for the appropriate technique to deal with the harmful spirit which has come into the sick man. Finally, in a dramatic climax, she literally seizes the offending spirit, likened to an object, expels it from his body, and then physically chases it out of the room with a broom through the smoke hole, with her colleagues on the roof confirming that it has indeed been routed. Then, the weight of the effort causes her to sink into unconsciousness. The shaman goes on expelling evil spirits until the patient is cured or the price becomes too high.

Finally, Zagoskin cannot resist the sly observation that these "doctors," not unlike the doctors of his own cultural tradition, seem to be more interested in etiology than therapy!

"The sick man is sitting in one of the corners in the winter house; he is suffering from a cold and in particular from rheumatic pains in the loins. Two lamps are burning in front of him, the back of his parka is pulled up over his head. Two shamans, one on each side of him, are singing from time to time and beating on drums. Behind can be seen the dishevelled head of an old woman: she is cawing like a crow, nodding at the back of the sick man, and conferring with somebody or other. When she runs into difficulties or sees that she is not meeting with success she changes her voice, chatters like a magpie, barks like a dog, and, finally, she sets to howling like a wolf and attacking the sick man as though she was biting at his back. She seems to tear something out of him and throw it into the air. The drums beat louder. Then the old woman jumps up, strips the parka from the patient, and sets to shaking it. Then she seizes a broom and starts gesturing with it in all directions; she sweeps under the benches as if she were chasing somebody out. They open the skylight. Four other shamans on the roof of the house strike their drums and shout: 'He has run away! He has run away! Oo! Oo!' The old woman either pretends to faint or actually loses consciousness. This is the third spirit she has expelled from the sick man; how many remain in him there is no telling. If this is the last one, the patient should soon recover; if not, the shamanizing continues as long as he wishes, presumably, since he is the one who pays for it. Sometimes the price is very high. The shamans affirm that their whole skill consists in discovering which spirit has settled in the sick person; they do not consider that expelling the spirit is important. Is it not the same in medicine?

"The time advances towards midnight; the natives settle to sleep, and when all is quiet the husbands leave [the kazhim] to join their better halves."

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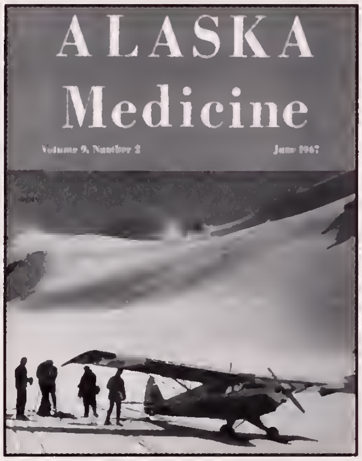
From Out of the Past...

Gloria Park, M.D.



Volume 9 , Number 2 "Raven Glacier"

Volume 9, Number 3 "Matanuska Glacier" 1996

Note new format above of *Alaska Medicine* in 1967 and the following review of the history of the journal by Dr. Betsy Tower:

Through the Retrospectiscope

by Elizabeth A. Tower, M.D.
former editor

ALASKA MEDICINE came into being on the crest of the enthusiasm over statehood in 1958 and 1959. In May of 1958 at the last Territorial Medical Association meeting in Fairbanks, a committee was appointed to investigate the feasibility of publication of an Alaskan medical journal. At the first meeting of the Alaska State Medical Association in June of 1959, the committee report was presented in the form of the Vol I, No. I (March 1959) issue of *Alaska Medicine*. In the forward to the first edition, the Editor-In-Chief, Dr. William J. Mills, Jr., Anchorage orthopedic surgeon, waxed poetic in proclaiming "we are given the opportunity now to insure that no longer need the unrecorded medical voice be sent plaintively wailing across the waters of the Inland Passage, to quiver in the Susitna Flats, or be sent rumbling toward the peaks of Mt. McKinley, only to wither and be dissipated in the wilderness."

Withe the enthusiastic acceptance of this committee report, the "Rubicon was crossed" and *ALASKA MEDICINE* came into being. The second edition carried letters from Herbert Hartley, M.D., Editor of *Northwest Medicine,* with whom Alaska had previously been affiliated, congratulating *ALASKA MEDICINE* for having sprung "Like Athena — complete in every detail"; from Joseph Garland, M.D., Editor of the *New England Journal of Medicine,* who honored *ALASKA MEDICINE's* birth in an editorial in his revered journal; from Johnson F. Hammond, M.D., Editor of the JAMA; and even from Bradford Washburn, Director of the Boston Museum of Science, informing the editorial staff that his photograph used on the cover of the first edition was the North Face of Mt. Brooks, not McKinley as advertised.

In the early years Anchorage internists William O. Maddock, Robert D. Whaley, and Rodman Wilson served as working Editors. Classic articles on such Arctic oddities as botulism in muktuk, salmonella in sea gulls, and climbing Mt. McKinley, were plentiful; eye catching photographs of Alaskan scenes were contributed by physician photographers; and drug companies were eager to finance the journal through their advertising despite the limited circulation which resulted in as many copies of each issue going as exchange journals to other states as to any other category of subscriber. Exchange journals from other states flooded the editor's office, were projected as the basis for a State Medical Library, and eventually ended up stored in an antiquated two room log cabin.

In 1961, I took over the management and editing of *ALASKA MEDICINE,* complete with cabin full of its exchange journals, and continued to bear these dual responsibilities until 1966 when the office of the Alaska Medical Association was established in Anchorage with a full time Executive Secretary. Throughout these five years, *ALASKA MEDICINE* has always come out, often two months late and frequently undernourished both in scientific content and in advertising, but nevertheless it has come out and has weathered the storms of a bankrupt printing house, a decrease in national advertising by drug companies, an earthquake and tidal wave. (The latter disaster was capitalized on with a special Earthquake Edition, which was published in June 1964, and was in such demand by civil defense agencies through the country that an additional special printing was required.)

Throughout the years the journal has been graced by articles featuring the history of medicine from the days of Steller and Bering (March 1967) through those of the Russian America Company (June and September 1962) and the Dogteam Doctor J. H. Romig (December 1963) to the present and by an informative and amusing gossip column "Muktuk Morsels" compiled until the past year by Dr. Helen Whaley.

Dr. Arndt von Hippel has now taken over the responsibilities of editorship and already the infusion of new blood is evident in the increased scope of the magazine. The business management carried o by Robert Ogden, the Executive Secretary of ASMA, is on a firm foundation. Good days should be ahead for *ALASKA MEDICINE*, but as before, the magazine will only be as good as the quantity and quality of the contributions by the physicians of Alaska. If they do not wish to have "the unrecorded medical voice . . . sent plaintively wailing across the waters of the Inland Passage. . . ", they should commit it to writing and submit it to *ALASKA MEDICINE!*

Another historical milepost:

NEW CARDIAC CATHETERIZATION LAB

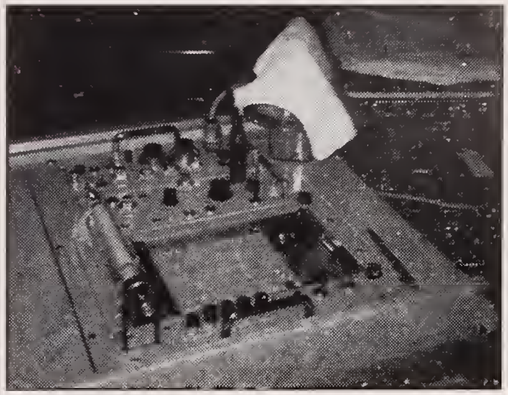
by Frederick R. Hood, M.D.
and Arndt von Hippel, M.D.

The cardiac catheterization laboratory at the Providence Hospital has now been in operation for about six months. Equipment includes an image intensifier, a rapid film changer and pressure injector for angiocardiography, sanborn pressure measuring and recording equipment, and Instrumentation Laboratory equipment for pH and gas analysis.

Diagnostic or preoperative studies have been done on patients with mitral or aortic valvular disease, patent ductus arteriosus, and atrial septal defect. In several cases surgery was found unnecessary or performed elsewhere on the basis of these studies. The patient with the patent ductus arteriosus had his diagnosis established and repair performed in Anchorage.

In the next year we hope to install Cine or T-V video equipment. This will finally make coronary arteriography available in Alaska. Coronary artery visualization for localization of the ischemic area is necessary before surgery is considered. Effective procedures are available for myocardial revascularization, in particular internal mammary implantation, and can now be performed in Alaska.

Coronary angiography has been indispensable in evaluating the efficacy of the various surgical approaches to coronary artery disease. It has also been of assistance in the diagnosis of coronary artery disease by providing a source of objective data in addition to the ECG. Frequently Cine coronary arteriography can establish the presence of significant coronary disease. On the other hand normal arteriographic findings offer substantial support for some other etiology of the symptoms.



And another milepost in *ALASKA MEDICINE:*

TENTH ANNUAL LEDERLE SYMPOSIUM FOR ANCHORAGE

A decade of annual postgraduate sessions for Alaska physicians was observed on February 25, 1067, when the tenth annual medical symposium sponsored by the Anchorage Medical Society and Alaska Chapter of the American Academy of General Practice was conducted at the Anchorage Westward Hotel.

Made possible by a grant from Lederle Laboratories, the Anchorage symposia have attracted a total attendance o 938 physicians from throughout the state.

Gerald Egelston, manager of educational services for Lederle Laboratories, reported that Anchorage, because of its size and location, is the only city in the nation in which the symposiums have been repeated for ten successive years. Attendance is acceptable for six hours of continuation study credit by the American Academy of General Practice.

The first five Anchorage meetings were designated as "post graduate seminars with round table discussions." Since 1963, each of the annual meetings has been identified as a "Symposium."

Program chairman for the first postgraduate session in 1958 was Dr. George Hale. Medical educators traveling to Alaska for the initial meting were Dr. Charles Bailey, Professor of Thoracic surgery at Hahnemann Medical Center, Philadelphia; Dr. Fred M. Taylor, Associate Professor of Pediatrics, Baylor University College of Medicine, Houston; Dr. Walter S. Priest, Associate Professor of Medicine, Northwestern University Medical School, Chicago, and former president of the American Heart Association.

Moderators for the pioneering session were Anchorage Doctors Francis J. Phillips, John C. Tower and Robert B. Wilkins. Attendance at the first session was 77 physicians.

Program chairman for the 1967 symposium was Dr. Frederick J. Hillman. Visiting speakers were Dr. Matthew Block, Professor of Medicine McGill University Faculty of Medicine Montreal. Moderators for the tenth symposium were Doctors Frederick R. Hood and Winthrop Fish.

Muktuk Morsels by Arndt von Hippel, MD — Dec '66 - Feb '67

Anchorage Medical News has been overshadowed by the sudden and untimely death of Dr. Les Margetts. In his 12 years of active surgical practice in Anchorage Les had become a loved and respected community figure.

Dr. William Mills was awarded the Bronze Star for bravery in the diagnosis and treatment of a South Vietnamese marine with a huge subcutaneous mass of recent onset. Because of his language problem, the patient kept thumping the mass, which absorbed Bill's attention completely when he discovered that it was a live mortar shell. Apparently on his way to South Vietnam for a six month tour is Dr. Alan Homay. We expect more information shortly. Dr. Marcell Jackson has resigned from the Doctors Clinic and has taken over Dr. Homay's practice.

Dr. Donald R. Rogers of Seattle, a board qualified pathologist, has joined the Doctors Clinic in Anchorage. Dr. Merritt Starr has taken over the State Medical Society T-V program series. Dr. Elizabeth Tower has resigned as Editor-in-chief of Alaska Medicine after many productive years of labor on the editorial rock pile. The many duties that she so ably performed will be divided between the new editor and Mr. Bob Ogden. Mr. Ogden is the new Alaska State Medical Society executive secretary. He was most recently the business manager of the Juneau Clinic and chairman of the Juneau Hospital Committee.

Dr. Grace Jansen recently was married to Vin Hoeman, a well known American mountaineer whose ideas of comfort are off by about 100 degrees (Fahrenheit). Dr. Bruce Wright recently married Nancy Allison of Anchorage. Drs. Fred Hillman and Louise Ormond Hillman have adopted an infant son. Dr. Richard Paul had his second son (third child) and Dr. Paul Dittrich recently had his first child, a boy.

We hear that Drs. Robert and Helen Whaley are reserving their decision on returning to Anchorage pending clarification of medical liability issues and legislation. Many Alaskan physicians share their doubts on continuing medical practice in Alaska under present circumstances.

As February ends Dr. George Wichman is reported above 17,000 feet with a group of mountaineers making the first winter ascent ever attempted of Mt. McKinley. We will have more on this later.

Dr. Harold Bartko has gone to Nome from Anchorage and will work at the Maynard McDougall Memorial Hospital. At present he is the only physician in Nome. He was recently appointed to the Alaska State Health Facilities Advisory Council by Governor Nickel.

From Seldovia we learn of the recent death of Mrs. Florence Armstrong. Dr. O. H. Armstrong is reported out of the state at present. Dr. John Fenger of Homer is taking a two-year residency in Physical Medicine and Rehabilitation at the University of Washington. (P. O. Box 758, Bothell, Washington) Dr. Joseph Deisher visited Seward in December and then returned to the Marshall Islands for the last months of his tour there. Dr. Deisher plans to take a fellowship at the University of Illinois in Continuing Medical Education as preparation for a position as Director of Medical Education.

Dr. Bob Johnson of Kodiak was appointed to the

Alaska Board of Basic Sciences. Dr. David Sammann left Skagway in December for graduate work at the University of Washington. He expects to return in April.

Dr. Joseph Rude has retired from the Doctors Clinic in Juneau. His son, Dr. Donald Rude, passed his Boards in surgery and is now practicing in Swahili at the Lutheran Hospital, Kiomboi, Tanzania, Africa.

Dr. John Beeson (Alaska Medical License #20, 1916) who practiced in Anchorage many years as the chief surgeon of The Alaska Railroad, before moving to Ketchikan, is hale, hearty and retired in La Jolla at age 94. In 1933 Dr. Beeson moved to Wooster, Ohio and founded a medical clinic there, staffed originally with his two sons; Dr. Harold Beeson, a former Anchorage Times reporter and now Assistant Medical Director for the Department of State in Washington, and Dr. Paul Beeson, now the Nuffield Professor of Medicine at Oxford University in England.

Dr. **Henry Wilde,** formerly of Juneau, is now regional health officer for the Foreign Service Diplomatic Corps in Guinea, Senegal and Mauretania. Dr. Wilde is stationed in Conakry, Guinea.

With the advent of the new Republican administration Dr. Levi Browning has been replaced as Commissioner of Health and Welfare by Dr. Wallace J. Chapman.

(continued from page 141 - Grace Akiyama)

She was always very generous, sharing her many talents with friends and organizations, her family wrote.

She is preceded in death by her parents.

Survivors include her husband of 44 years; son, Alan Kent; daughter and son-in-law, Lisa Anne and Gary Crowe; grandsons, Drew Curtis and Brett Arthur McCurley, all of Juneau, sister and brother-in-law, Dr. May M. Ebihara Gelfand and Marvin Gelfand; and nephews, Adam and Jeremy Gelfand, all of New York.

Obituary dated August 27, 1996, reprinted with permission from the Juneau Empire .

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